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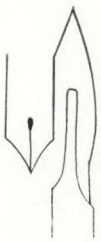
Volume 16, issue 3

Canadian Medical Association



QUILL ON SCALPEL This section provides a medium through which Canadian surgeons can declare themselves, briefly and informally, on the day-to-day affairs of surgery.

PROPHYLACTIC ANTIBIOTICS

The use of antibiotics to prevent surgical infections fell into disrepute 10 or 15 years ago when it became obvious that the dictum "prophylactic antibiotics might do some good and cannot do harm" was totally wrong. The establishment of the use of antibiotics to prevent wound and other infections associated with surgical operations has awaited carefully performed, prospective, randomized trials on patients. Several excellent trials have now confirmed the value of prophylactic antibiotics in surgical practice. The question now is not whether they should be used but rather in what situation, for how long, in what dosage and by what route.

The reasons for not using antibiotics indiscriminately are: (1) the failure to demonstrate any decrease in infections, (2) the development of antibiotic-resistant organisms, (3) adverse drug reactions, (4) the masking effect on undrained abscesses and (5) the cost.

How should we use antibiotics preventively? The following conditions should be satisfied: (1) the antibiotic should be effective against the particular organisms likely to be encountered during the operation; (2) the antibiotic should be present in the wound in high concentration at the time of incision; (3) a short-term (one to three days), high concentration, low toxicity course should be used; and (4) the operation should carry a significant risk of contamination and wound infection.

In which operations should we use prophylactic antibiotics? In *clean* operations, for example hernia repair, varicose-vein stripping, thyroidectomy and mastectomy, there is no evidence that prophylactic antibiotics are of any value. Many surgeons have a wound infection rate of less than 1% for such cases without the use of antibiotics.

A *clean-contaminated* operation is one in which the gastrointestinal, respiratory or genitourinary tract is entered but without significant spillage. In this type of wound and in *contaminated* wounds in which there is gross spillage, or if acute inflammation is encountered or in a fresh traumatic wound the value of prophylactic antibiotics is much clearer.

A *dirty* wound is defined as one in which pus is encountered or a perforated viscus exists. The use of antibiotics in such a case is therapeutic rather than prophylactic.

What is the evidence that prophylactic antibiotics are of benefit in clean-contaminated and contaminated operations? The results of several double-blind, randomized trials are available. Polk and Lopez-Mayor[1] performed a randomized, double-blind prospective trial of consecutive patients undergoing elective operations on the gastrointestinal tract exclusive of the gallbladder. The control group received a placebo; the experimental group received cephaloridine 1 g. intramuscularly immediately before being taken to the operating suite, and at five hours and 12 hours postoperatively. Serum antibiotic activity was assayed in all patients at one, five and 30 hours. Wound antibiotic level was determined at the time of

wound closure. Intestinal preparatory antimicrobial agents were not used and 104 of the 250 patients in the study had colorectal operations. There was a highly significant reduction in wound and intra-abdominal infection rates in the patients on the antibiotic regimen. The infection rate for the patients who received only a placebo was 29% and for the group who were given cephaloridine it was 6%. If colorectal operations were considered as a separate group the infection rates were 30% and 8% respectively.

Bernard and Cole[2] randomized the use of penicillin, methicillin and chloramphenicol (the antibiotic group) with administration of a placebo in 189 patients undergoing elective operations on the gastrointestinal tract including biliary surgery. The infection rate was 27% in the placebo group and 8% in the antibiotic group.

Andersen, Korner and Østergaard[3] in an amazing study demonstrated the value of topical antibiotics in colorectal surgery. The series comprised 240 patients who had colonic or abdominoperineal resections of the rectum. All patients had mechanical preparation of the colon. With the exception of 55 patients with low rectal carcinomas all patients also received intestinal antibiotics—neomycin sulfate and bacitracin 1.5 g. every six hours and of chlorchinaldole 100 mg. every eight hours for three days before operation. In the treatment group ampicillin 1 g. as a powder was applied to the subfascial and subcutaneous spaces. Wound infection was defined as accumulation of pus occurring within 30 days of operation and requiring surgical revision. The results were highly significant; the wound infection rate in controls was 18%, in the ampicillin group it was 2.5%.

Oral antibiotics as preparation for colon surgery appear to have lowered the infection rate from about 50% to 2%[4]. Somethinng further is needed.

Why not use delayed primary wound closure in all cases of clean-contaminated and contaminated operations? This technique is not without morbidity. One would prefer primary closure if primary healing is a certainty or may be expected in 95% to 97% of patients.

A guideline for use of prophylactic antibiotics and wound closure might be the following: (1) Clean operation—no antibiotics, primary wound closure. (2) Clean-contaminated operation—prophylactic antibiotics, primary wound closure. (3) Contaminated operation—prophylactic antibiotics, delayed primary wound closure. If the results of Andersen, Korner and Østergaard[3], using combinations of oral, systemic and local antibiotics can be duplicated, it is possible that primary closure is advisable in contaminated operations as well. (4) Dirty cases—antibiotics for treatment and delayed primary or secondary wound closure.

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REVIEW ARTICLE

IMPLANT MATERIALS AND THEIR USE IN ORTHOPEDIC SURGERY

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THE first known use of metallic implants introduced into the human body for the treatment of disease dates back to 2700 B.C., when the ancient Chinese used a form of treatment known as acupuncture.[1] This was not a method of gaining structural stability, but consisted of inserting fine needles of silver or gold into tissues at the site of pain or at a distant site. The original text on acupuncture, published some 2300 years ago, described 12 channels or "meridians" in the body. Insertion of a needle at any of about 365 positions along these channels was thought to have a physiologic effect. The needles, which were ½ to 10 inches long, were allowed to remain in place from a few minutes to several days, with varying effect. Numerous forms of this treatment are still used in some parts of the world, particularly in China, where scientific acupuncturists, using stainless steel needles, have added about 200 points to the original positions of insertion.

After the introduction of acupuncture, little progress was made in the use of metal implants for many hundreds of years, until Lusitanus, in 1560, devised a gold plate for the repair of cleft palate defects.[2] The following years evoked great turmoil over the use of internal fixation. In the early 1770s surgeons began to use various types of metal wire to secure fracture fragments. However, during this pre-microbiological era, surgical procedures were frequently complicated by the development of gangrene which was observed to "kill patients like a pistol shot".[1] Many surgeons believed that the implant materials were directly responsible for this infection. Pancoast, in his famous treatise on operative surgery (1844),[3] finally concluded that metal implants "should not be used in fractures, as they cause bone necrosis".

Malgaigne,[4] in 1847, proposed a compromise to the opposing views of open versus closed reduction. He introduced an external-internal fixation unit for fractures of the knee and elbow, in which adjustable hooks were attached to a clamp. The hooks went through the skin into the bone fragments and the external clamp was tightened using a special wrench. Although this particular device had only short-lived success, orthopedic surgeons currently use modifications of this procedure, e.g. the Roger-Anderson apparatus.

In the second half of the nineteenth century two extremely significant developments pushed orthopedic surgery toward the modern era. In 1860 Lister[5] recognized that infections were caused by micro-organisms and advocated antiseptic precautions in surgery. In 1895 Roentgen's monumental discovery[6] enabled orthopedic surgeons to view the reduction being obtained and precipitated a revival of interest in open reduction and internal fixation of fractures.

In 1886 Hansmann[7] devised the first practical metal bone plates and screws. The screws and one end of the plate were left protruding from the wound and subsequently could be removed easily using a watch key. Lane[8] further developed the concept of plates and screws and introduced a rigid plate which could be permanently left inside the wound (Fig. 1). His greatest contribution, however, was his insistence upon rigid asepsis which was later known as the "Lane no-touch technique". He claimed never to have observed "rarefying osteitis" in any of his patients, and stated emphatically "rarefying osteitis in plain English means dirty surgery and is a useful term to cover surgical incompetence".[1]

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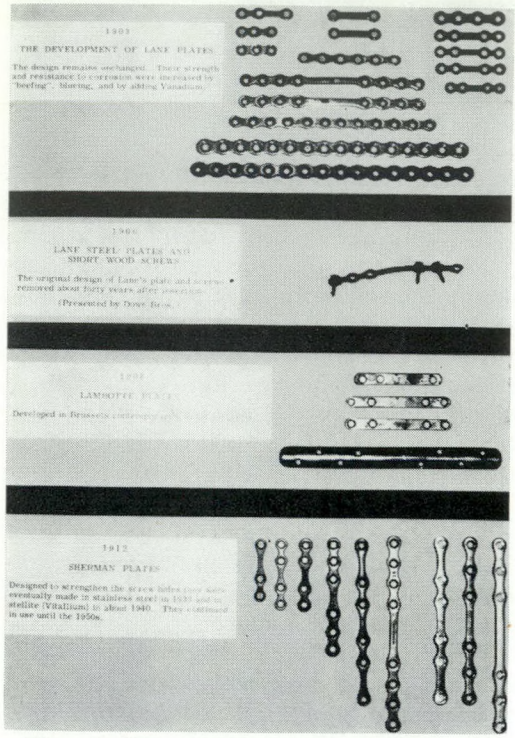

Fig. 1.—The development of metal bone plates and screws. (Courtesy of the Bristol Royal Infirmary, Bristol, England.)

In the following years, with greater insistence upon aseptic precautions, significant advances were made in the use of internal devices for the fixation of open fractures. Sherman observed that the Lane plate, which was made of tool steel or crucible steel, was too brittle to withstand bending or twisting strains. In 1912, after three years of research, he developed a new steel alloy, which contained vanadium and chromium and lower levels of carbon (Fig. 1).[9] Bone plates made of this vanadium steel were three times stronger than other forms of steel. He subsequently used self-tapping, vanadium-steel, bone screws and recommended laterally curved plates which introduces less metal into the wound (Fig. 1).

In 1913 Hey-Groves[10] conducted a study of tissue tolerance to various metals and advocated the use of nickel-plated steel. From 1912 to 1914 numerous workers began to use metallic bands to secure fractures. Increasing experimentation led to tremendous controversy, because metal bands which were left in place produced circumferential bone notching that frequently resulted in another fracture. This problem was solved in 1913 by Parham and Martin[11] who developed a removable band.

The original techniques of intramedullary nailing were first developed by Hey-Groves in 1908 for fractures of the shaft of long bones and of the neck of the femur.[12] He used various types of material, including beef bone, ivory pegs and several different metal alloys (Fig. 2). Many problems developed—the organic materials frequently loosened and failed and the steel implants often corroded. As more workers experimented with a wider variety of new metals, adverse chemical tissue reactions to these materials became increasingly apparent. In 1927 Lambotte described the "electrolytic phenomena" associated with certain metallic appliances in the body,[1] and workers began to experiment with still other metal alloys.

In 1931, with the development of stainless steel, Smith-Peterson, Cave and Vangorder[13] reintroduced the procedure of hip nailing. In 1936 Rush and Rush introduced intramedullary pins with a curved extraction hook (Fig. 2) to facilitate their subsequent removal.

In the following year surgeons became increasingly aware of the mechanical forces acting at different fracture sites and created a wide variety of implant designs. Although intramedullary internal fixation had been attempted previously, Küntscher[14] in 1940 became the first to develop successfully this technique. He used V-shaped nails of stainless steel which were driven down the medullary cavity of a fractured bone. In 1943 he changed the cross-sectional design to a "clover leaf" to increase stability by filling the medullary canal (Fig. 2); thus the mechanical forces of angulation, lateral displacement and torque were better controlled. Motion of the adjacent joints could be initiated immediately and muscle atrophy and stiffness were

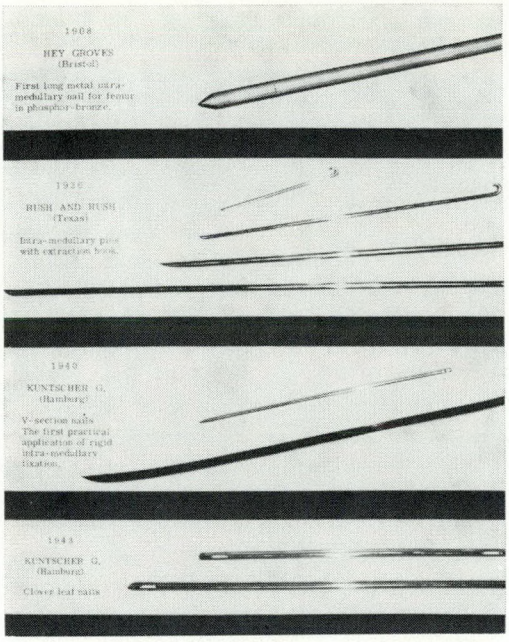

Fig. 2.—The development of intramedullary fixation devices. (Courtesy of the Bristol Royal Infirmary, Bristol, England.)

minimized. In some fractures of the femur, union was so rapid that patients could walk without additional fixation in two to three weeks. Although Küntscher originally recommended his method for fractures of all long bones, intramedullary nailing of thousands of fractures over the following decade showed that, except for those of the femur and other selected cases, most fractures could be better treated by other measures.[15]

The modern era of metallic implants has been characterized by the development of more acceptable alloys and by advances in the mechanical engineering of prosthetic appliances. In 1936 a new alloy of cobalt, chromium and molybdenum (vitallium) was shown to be essentially inert in body fluids.[16] To date, over 15 million vitallium appliances have been surgically implanted. Newer minimally-reactive stainless steels have also been developed. With the development of image intensification in the 1950s, reduction and internal fixation of fractures could be observed readily without subjecting patients to high doses of radiation. Modern implants have now been improved to the point where they are used with almost complete confidence by orthopedic surgeons.

During the past decade, Müller, Allgöwer and Willenegger in Switzerland[17] have developed numerous highly refined techniques for applying compression devices to rigidly fixed and perfectly reduced shaft fractures. The principle underlying this procedure is that rigid fixation of bone fragments under compression increases the area of contact and minimizes shearing and torsional forces. There is no resorption of fragments, but primary bony union occurs in the absence of visible endosteal or parosteal callus. Although the Swiss group have described carefully over 14,000 operative cases in which compression plating has been used[17] and have demonstrated many advantages of this technique, it remains to be proved that compression in itself accelerates normal fracture healing in cortical bone. Research workers, however, are currently studying this relatively new concept of internal fixation.

Cementing Substances

In recent years, concurrently with the search for the ideal material for metallic implants, research workers have explored the possible use of cementing substances in orthopedic surgery. Three potential uses for bone cements are: (1) to function as space fillers in cancellous bone defects, (2) to stabilize fractures and (3) to secure metal implants to bone. Each of these situations requires a substance with specific properties.

Cementing Substances to Fill Bone Defects

The ideal space-occupying substance in bone should: (a) be easily sterilized; (b) have the required physical properties of strength, elasticity and hardness; (c) be easily shaped before setting; and (d) be biologically inert. One of the first materials so tested

was plaster of Paris, which, when mixed with water, becomes $CaSO_4.2H_2O$ (gypsum). Since the initial investigations in this area by Trendelenburg in 1892, hundreds of workers have attempted to use this substance in bone defects, with only limited success.[18] The largest obstacle to its use is the effect blood and tissue fluid have on its setting. Under selected conditions, however, it has been observed that plaster of Paris is resorbed rapidly with osseous regeneration.

During the past decade, extensive research has been done on polymeric adhesive materials, such as the cyanoacrylates, polyurethane, and various epoxy resins. Although these polymers were originally thought to be chemically stable and biologically inert, subsequent investigations have, in many instances, raised serious doubts about their stability and toxic properties.[19, 20]

During the past few years, implantation research has been directed towards the development of a self-locking implant either (a) hydrophilic materials capable of expansion on setting or (b) porous substances into which bone would grow to provide stability. Many hydrophilic acrylate polymers ("hydrons", "hydrogels", e.g. 2-hydroxyethyl methacrylate) appear to possess a self-locking capacity by virtue of their ability to absorb fluid with concomitant swelling.[21] Hydrons of high mechanical strength are obtained if their polymerization is carried out under conditions which provide a water content of 45%. These gels can, however, be polymerized at a lower water content (e.g. 25%), to provide a solid partially pre-swollen material, which on implantation swells to provide a self-locking condition. The hardness or resiliency of these gels can be controlled by the proportion of cross-linking agent used in polymerization.[21] Some of these compounds appear to be biocompatible and will undoubtedly be developed further for use in orthopedic surgery, possibly to replace focal defects in articular cartilage. A major disadvantage is their friability or lack of tolerance to major stress—a problem which may be solved by further research on their chemical structure.

Polyvinyl and acrylate-amide "sponges" were studied initially as potential porous implants, but their poor physical properties reportedly limited their use.[22] Recently, as an extension of the space-age interest in heat-resistant substances, the search for a stronger material led to the development of porous ceramic substances.[23] The initiation of ceramic implant programs in 1968 led to their use as solid wedges and spacers in osteotomy and limb-lengthening procedures. Further experimental applications of these materials include total and partial hips and knees, temporomandibular condyles, mandible implants and cranial defect repair procedures. More recent experimentation in this field has led to the development of Cerosium,[23] a porous ceramic substance impregnated with an epoxy resin, which was formulated to have a pore structure and physical properties similar to bone.[24] This material will most certainly have further clinical applications in orthopedic surgery.

Other organic materials that have been used extensively in orthopedic surgery are preparations of silicone rubber (Silastic).[25] Silastic was originally introduced in 1966 by Howard as a pliable, one-piece, hinge-type, intramedullary prosthesis for metacarpophalangeal and interphalangeal joints. Following implantation the prosthetic stem glides freely within the intramedullary canal, or becomes totally or partially bound by a supportive fibrous joint capsule. Subsequent research has led to the development of radial and ulnar head, scaphoid, lunate, trapezium, and other prostheses. Recently a Dacron mesh has been applied to the external surface of the silicone finger-joint prostheses.[25] Fibrous tissue rapidly infiltrates this mesh to provide greater fixation of the prosthesis.

Stabilization of Fractures

Many cementing substances have been tested in an effort to provide

stability in certain pathologic and comminuted fractures of long bones. The ideal substance for this purpose should: (a) form a firm adhesion with bony surfaces, (b) not impede the normal fracture healing process, and (c) be subsequently resorbed without deleterious effect. Many of the previously mentioned polymeric compounds have been tested in this capacity, generally with poor results. One such material which appeared initially to satisfy the requirements of "non-toxic bone glue" was Ostamer (polyurethane).[26] This material was used extensively for two years in the United States, but it was subsequently abandoned because it failed to facilitate fracture union and produced a variety of side effects in susceptible patients.

Little progress has been made in the stabilization of fractures using cementing substances. Newer adhesives are undergoing clinical trials but none of these has yet reached the stage of widespread clinical application.

Cementing Substances to Secure Prostheses

The principal clinical success with cements has been achieved in the adhesion of orthopedic prostheses. Total hip prostheses are probably the most extensively studied internal prosthesis in orthopedic surgery. The concept of replacing a femoral head with a prosthesis was initially developed by Judet (using an acrylate plastic) in the late 1930s, and subsequently modified by Thompson, Moore, and others using steel and metal alloys.[27] Acetabular cups of Teflon (polytetrafluoroethylene), Teflon derivatives (Fluorosint), and high-molecular-weight polyethylene have been used. At present the most suitable materials for an artificial hip joint are vitallium or stainless steel for the prosthetic head, and high-density polyethylene for the acetabular cup.[27-29]

The basic concept of total hip arthroplasty involves resection of the femoral head and replacement with a long-stemmed prosthesis, which is mechanically fitted into the intramedullary canal of the upper end of the femur. The acetabulum is also replaced with a socket of polyethylene. However, under certain conditions, such as the presence of malfunctioning hip adductors or osteoporotic femoral shafts, the mechanical bond may loosen, with subsequent restriction of motion and joint pain.[29] Mechanical failure was thought to result from the abrupt transfer of stress from the rigid metal implant to the surrounding elastic bone tissue.

In the early 1950s surgeons began searching for a cementing substance with elastic properties intermediate between metal and bone, which could be interposed between the two surfaces. In 1958, on the advice of Smith, Charnley began to use methyl methacrylate for this purpose.[30]

Kiaer had previously used this acrylate polymer as early as 1941 in the fixation of the Judet femoral head prosthesis[31] but his early trials invariably failed because of the mechanical disadvantage inherent in the short-stemmed Judet prosthesis. Other workers[32] also used methyl methacrylate for femoral head replacements but did not insert it into the medullary canal.

Methyl methacrylate is a polymeric, self-curing, acrylate cement, which acts as a mechanical filler without having actual adhesive properties.[29] Its physical setting is facilitated by mixing prepolymerized acrylate, the monomeric form, and a chemical activator, which undergo an exothermic reaction to harden into a solid matrix in five to 10 minutes.

Immediately after mixing, the cement is forced down the femoral medullary canal. Insertion of the tapered prosthetic stem subsequently forces the cement into the cancellous lining of the marrow space to produce a cement thickness of 2 mm. to 4 mm. The prosthetic stem and the hardened cement sheath subsequently function as a single unit. Weight is initially transmitted through thousands of bony trabeculae in the endosteal femoral surface and subsequently through newly

formed fibrocartilage. Mechanical tests have shown a 200-fold improvement in load-bearing capacity of the prosthesis over a similar situation without cement.[29] Polyethylene acetabular cups are also firmly fixed with the acrylate cement.

To date, Charnley has performed more than 5500 total hip arthroplasties using methyl methacrylate with overall excellent results. Many patients with atrophy of the femoral cortex have demonstrated hypertrophy of this cortex after polyacrylate implantation, probably owing to the improved physiologic response to mechanical stress. However, the use of this cement is associated with two disadvantages: (a) heat production which causes some degree of bone death around the prosthetic stem, and (b) a mild cytotoxicity associated with the residual acrylate monomer in the set cement.[29] In spite of these disadvantages, methyl methacrylate has proved to be the most successful cementing substance used to date in total hip arthroplasties, and will undoubtedly be applied to other prosthetic replacement procedures.

The use of methyl methacrylate has not been confined to orthopedic surgery. Shapiro, in 1969, collected 480 references to the use of this acrylate polymer in other surgical specialties.[29] It has been used in plastic surgery to reconstruct the nose and orbital region, in dental surgery to secure both internal and external prostheses, in cranial operations to fill skull defects and to invest cranial aneurysms where standard techniques are not possible, in ophthalmic surgery for contact lenses, intraocular lenses, and to repair lacrimal ducts and, in otology to replace ossicles.

Further Developments in Bone Cements

In 1967, D. C. Smith, Professor of Dental Material Science at the University of Toronto, described the preparation and mechanical properties of a new dental cement, zinc polyacrylate (PAZ), composed of polyacrylic acid (Fig. 3) and a zinc oxide preparation.[33] The cement, he suggested, set because of a chemical bond between Zn^{+2} ions and four adjacent carboxyl groups in the polyacrylic acid molecule,[20] and produced a solid gel-like structure (Fig. 3). In addition to forming a strong mechanical bond, PAZ has the unique property of forming a tenacious chemical bond with suitably calcified surfaces through the chelation of calcium ions by free carboxyl groups in the acrylate polymer (Fig. 3).[34] PAZ was further shown to form a direct chemical bond with stainless steel and various other metals, facilitating its use as an adherent of implant materials to calcified surfaces.[35] A number of different chemical preparations of this polycarboxylate cement are currently being used in a variety of dental conditions.

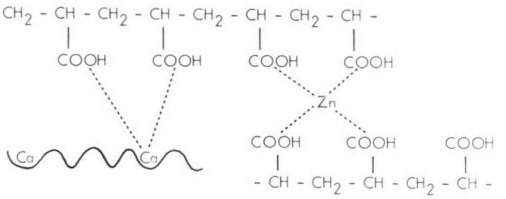

Fig. 3.—Postulated bonding mechanisms of polyacrylic acid to calcium and zinc ions.

Because of the unique binding properties of PAZ, and because in a preliminary clinical trial it appeared to be resorbed from bone without deleterious effect,[35] this polymer has been investigated as a potential implant material in orthopedic surgery.[36] During the past two years, in association with Dr. Smith, we have conducted experiments on the chemical, physical and toxicologic properties of PAZ as a bone cement.[36] This compound appears to possess many advantages as a potential implant material in orthopedic surgery. Unlike methyl methacrylate, its setting is not associated with a biologically destructive exothermic reaction. It has a high compression and tensile strength and these properties are subsequently improved by storage in water.[35] It is extremely stable and non-toxic *in vivo*

over prolonged periods of implantation.[36] Experiments are currently in progress to increase the binding properties of PAZ using different chemical preparations of the material. Experiments are also being conducted on the use of PAZ to secure prostheses to amputated limbs in experimental animals. Further investigation of polyacrylic acid and of various other biopolymers will undoubtedly lead to their ultimate use in many branches of orthopedic surgery.

Obstacles to the Development of Implant Materials

The development and ultimate successful use of implant materials in orthopedic surgery involve an integration of basic science and clinical medicine. Any material implanted into the body is continually subjected to numerous mechanical, chemical and metabolic forces. Over many years the substance is partially degraded into potentially toxic constituents. Any foreign material invariably causes some degree of chronic inflammation.

Before a material has been implanted into the body, the possibility of long-term malignant transformation must be evaluated with extreme care. At present there is little uniform agreement concerning the criteria of carcinogenicity. In the United States, materials must be accepted by the Federal Food and Drug Administration, whose criteria are presently ill defined. One problem with animal evaluation of implant materials is that the species in which a test is conducted may have a susceptibility to malignant transformation different from that of a human. Teflon, for example, was previously shown to be non-carcinogenic in animal implantation experiments and was therefore used extensively in orthopedic surgery. Subsequently it proved to be "one of the most irritating materials ever used in surgery, producing caseating granulomata, sterile pus, and bone erosion".[29] Conversely, methyl methacrylate was shown to produce malignant changes after implantation in rats,[37] but has failed to be carcinogenic after being implanted for as long as 20 years in human subjects.[5]

While the general lack of agreement on standards of implant quality and toxicity has resulted in the use of certain toxic materials in human subjects, it has also prevented the use of certain apparently non-toxic, potentially advantageous substances. Methyl methacrylate, for example, has long been used successfully and extensively in Europe and the United Kingdom but, only this year, has the Food and Drug Administration released it for use in the United States. This problem of induced toxicity must clearly be defined before progress can be made through implantation research.

Currently the development of cementing substances is directed towards materials which, in addition to having the required physical and chemical properties, would be totally metabolized in the body to non-toxic products. Methyl methacrylate, for example, which is only slightly degraded *in vivo*, appears to undergo two oxidation reactions to yield pyruvate, which is subsequently metabolized completely to carbon dioxide and water.[38] Many experiments are being conducted on naturally-occurring animal proteins which could be chemically hardened into implant substances. Continued basic and clinical research into biopolymers and other organic cementing substances will undoubtedly lead to the development of the ideal non-toxic biodegradable implant material for use in orthopedic surgery.

Summary

A review is presented of the historical development of metallic implant materials and cementing substances used in orthopedic surgery. Special emphasis is placed on recent advances in organic polymer adhesive materials and their biological advantages as implant substances. The current status and future goals of orthopedic implantation research are described.

The author wishes to thank Dr. R. W. Jackson, Director of Orthopedic Research, Banting Institute, for his enthusiasm, encouragement and valuable advice during the preparation of this paper.

The research conducted by the author and co-workers on polycarboxylate cements was supported by Medical Research Council of Canada Grant MA-2296.

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SURFACE AREA OF THE HAND AND DIGITS

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Vancouver, B.C.

IN 1967, Fairey and Cowan[1] reviewed the various techniques used in measuring body surface areas. Although the significance of the data so obtained is limited, the information has immediate practical application. By measuring the surface area of the hand and the digits the surgeon can obtain accurate data on which to base the size of free skin grafts and the design of pedicle flaps used in reconstructive procedures.

Benedict[2] in 1916, Weinbach[3] in 1938 and Geoghegan[4] in 1953 attempted to determine surface areas from photographs. More recently Hertzberg, Dupertuis and Emanuel[5] in 1957 and Beard[6] in 1967 developed a photographic technique that measures surface area accurately by constructing contour maps from stereophotographs of the body. Also, the surface area can be accurately determined by slope analysis,[7-9] but the equipment needed for this procedure is expensive and the calculations are time-consuming and involved. Computer programs have been written to calculate surface area from contour maps, and workers at the Medical Research Laboratories (A.F.S.C.), Wright-Patterson Air Force Base have made measurements by this method.[10]

Although stereophotogrammetric methods are more accurate, they cannot be used to measure the surface of the flexed hand. The present paper describes the results obtained in measuring the surface area of the hand in 10 subjects, volunteers from the staff and patients in the Department of Surgery, University of British Columbia. In this technique the skin surface is coated with liquid Silastic which polymerizes under the influence of a catalyst. The maximum error with this method was 3.2%.

MATERIALS AND METHODS

The technique described by Fairey and Cowan[1] for measuring the surface area of the face was modified for the hand. The catalyzed liquid Silastic is spread thinly over the entire hand and carried about one-half inch proximal to the wrist line.[11] Several applications are required. The position of the hand is maintained carefully until the Silastic polymerizes. All three positions (standard, flexion and extension) can be moulded in approximately 2½ hours. The mould is then cut from the hand with scissors (Fig. 1) and allowed to dry thoroughly. For identification purposes the position and the regions of the hand are marked on the mould in Bonney's blue dye. The mould is then trimmed and cut in the various regions with a scalpel. The edges of each section

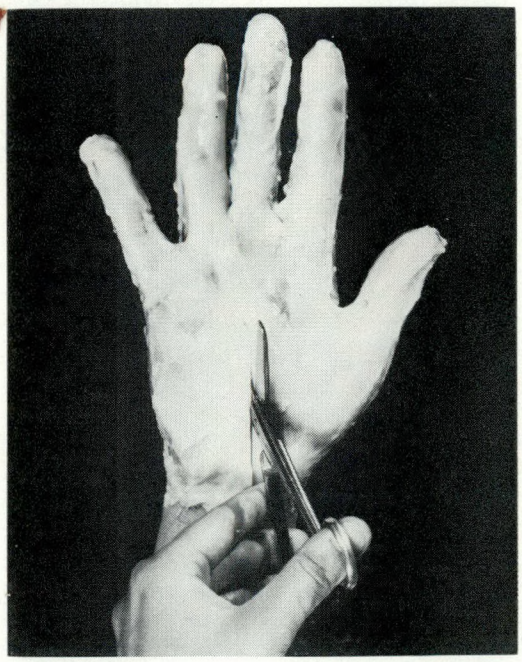

Fig. 1.—Incising the silicone moulage.

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Presented at the meeting of the Canadian Society of Plastic Surgeons, Jasper, Alta., June 1971.

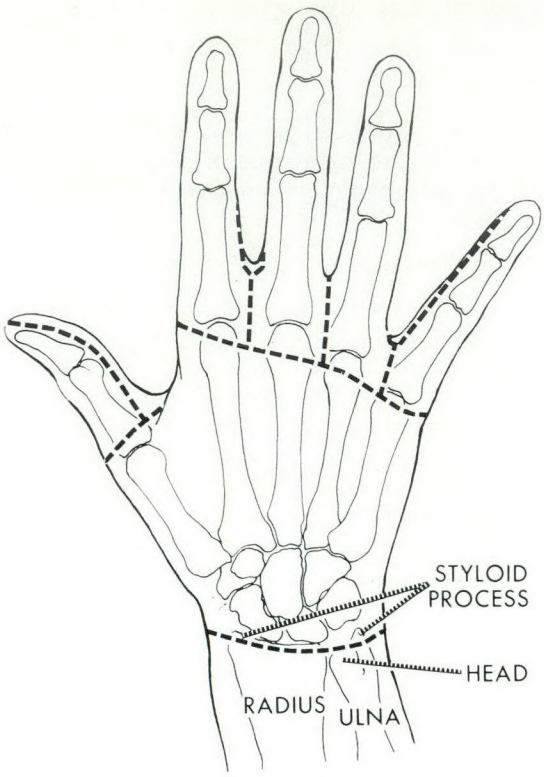

Fig. 2a

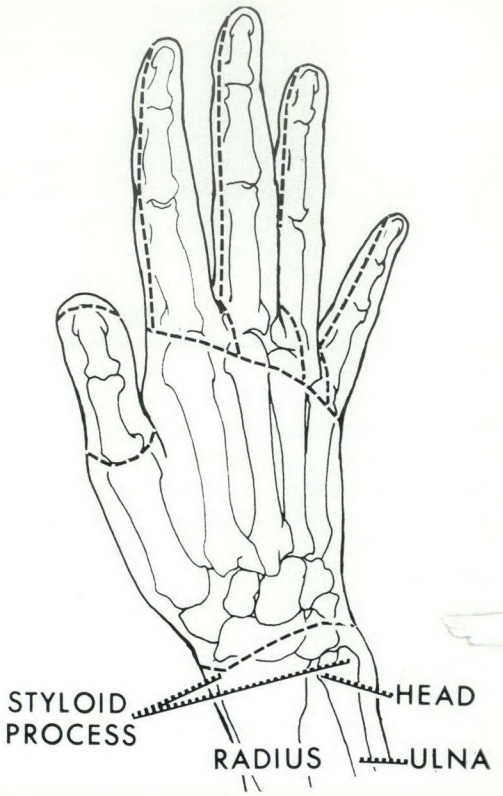

Fig. 2b

Fig. 2.—Surface markings to illustrate the boundaries of the dorsum of the hand and the digits: (a) dorsal, (b) dorsolateral.

are trimmed and shaved to a thickness of approximately 1/16 inch. This is especially necessary on the cylindrical pieces because the Xerox machine copies only the outside edge. The pieces are laid on the glass plate of the Xerox copier with the inner (skin) surface next to the glass. A black poster card is placed over the pieces after a check is made for holes or overlap. The pieces are flattened completely with the Xerox cover and photocopied. The prints are again checked for overlap and each piece is identified and numbered. Lastly, we measure the Silastic prints with a planimeter and calculate the area of the hand and its regions.

Mapping the Hand

On a skeleton the boundaries of the hand and the digits are readily apparent, but it is difficult to define the surface markings on a living subject. Anatomy textbooks do not precisely limit the boundaries of the hand, fingers or the thumb. For the purposes of this study we chose the radiocarpal joint as the obvious proximal limit of the hand. When the palm and forearm are resting on a horizontal surface (Fig. 2), the closest approximation to this joint is the straight line that connects the following points: the tip of the radial styloid, the tip of the ulnar styloid (easily found in full pronation) and the distal margin of the head of the ulna.

The proximal limits of the fingers were represented on the ventral surface of the hand by a line (Fig. 3) drawn half way between the proximal flexion crease of the fingers and the distal and middle palmar flexion creases. The latter was made into a single line by connecting the medial end of the distal palmar crease to the lateral end of the common crease of the thenar and middle palmar creases. As Fig. 4 illustrates,

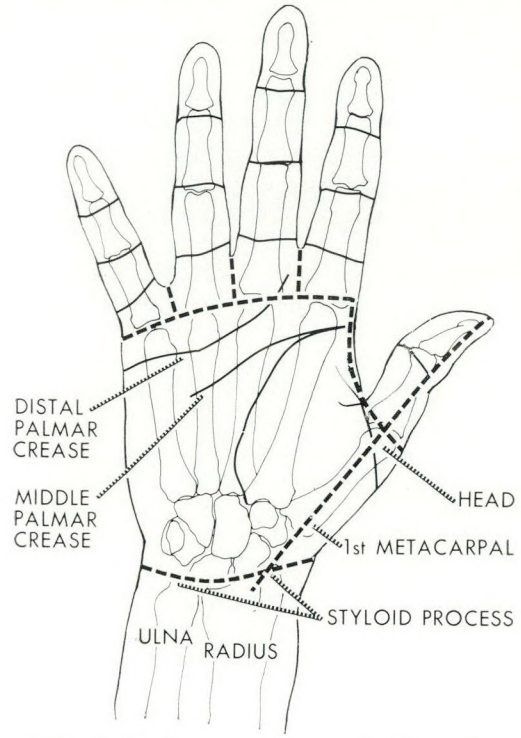

Fig. 3.—Surface markings of the palmar surface of the hands and digits.

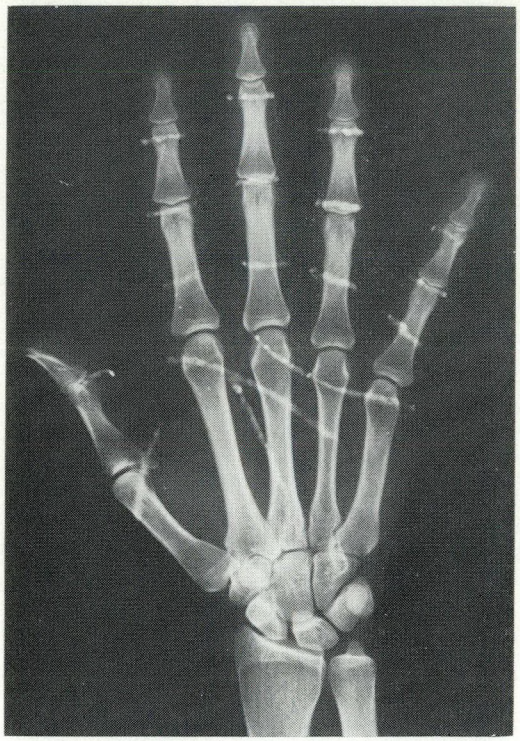

Fig. 4.—Barium has been painted on the palmar skin creases to illustrate the relationship of the thenar, mid-palmar, distal palmar and the digital skin creases to the metacarpophalangeal and interphalangeal joints.

this marking was a compromise, because the metacarpophalangeal joints are distal to this marking. The metacarpophalangeal joint of the index finger is almost midway between the proximal finger crease and the mid-palmar crease, whereas the metacarpophalangeal joints of the medial three digits are about one-third of the distance from the distal crease to the proximal finger crease. This compromise with respect to the boundaries of the fingers and palm incorporates part of the palm in the finger measurements. We used the line that joins the prominence of the metacarpal heads, and is evident on flexion of the fingers, as a dorsal marking on the proximal end of the fingers. The fingers are separated by straight lines midway between the metacarpophalangeal heads at the base of the proximal phalanges of the dorsal and the palmar surface.

To represent the medial boundary of the palm a line was drawn from a point between the pisiform and the triquetrum to the midpoint of the medial side of the little finger (with the hand and the forearm maintained in the same horizontal plane). For the lateral boundary of the palm a straight line was drawn from the medial side of the prominence of the lateral surface of the lower end of the radius and a little above the styloid process (this prominence receives the insertion of the brachioradialis tendon) to the midpoint of the lateral side of the thumb where it intercepts the dorsal and the proximal limits of the thumb. (The palm and the forearm were maintained in the same horizontal plane with the tip of the thumb opposed to the base of the little finger.)

The dorsal and the ventral boundaries of the palm and the dorsum of the hand are completed by drawing a line from the intersection of the proximal limit of the index finger and the lateral dorsal-ventral boundary, along

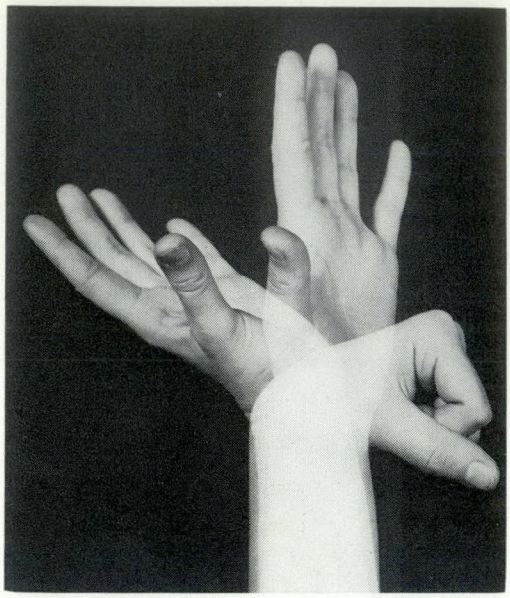

Fig. 5.—Moulages of the hand were made in three illustrated positions, *viz.* full extension, standard or mid-position, and full flexion.

the interdigital fold between the first and the second metacarpals to the proximal boundary of the thumb.

Position of the Hand

In determining the surface area we studied three positions of the hand (Fig. 5): (1) The position of flexion, obtained by flexing the fingers, then flexing the wrist with the thumb overlapping the adjacent two fingers; this produces the greatest expansion of the dorsal skin and the greatest contraction of the palmar skin. (2) Full extension at the wrist and fingers. (3) A standard position in which the pronated palm and forearm lie in the same horizontal plane with the fingers comfortably abducted.

Use of Radiographs in Mapping Lines and the Underlying Joints

We tried several different techniques to establish the relation between the mapping lines and the underlying joints:

(1) Stainless steel was taped over the mapping lines (the proximal limit of the hand, fingers and thumb in particular) and the hand was radiographed. The lines were very distinct but the tape immobilized the skin, giving an inaccurate result.

(2) Lead threads from surgical sponges were taped on the skin. They were more flexible than wire but, again, skin immobilization produced errors.

(3) Barium mixed with water was painted over the mapping lines. This eliminated the tape, but the barium dried and flaked off before radiographs could be taken.

(4) Barium was mixed with water and oil and applied to the hand with a pipette. Again the barium flaked.

(5) Finally, barium was mixed with oil only and applied with a pipette. By this means the barium remained in suspension in the oil, but the lines on the radiograph, although fairly wide, were not very distinct. This was the method used, for example, in Fig. 4. By the same technique, skin creases of the hand were made visible on the radiograph to show their relationship to the bones and joints.

Results

Using this method we measured the surface areas of the hands of the 10 volunteers. The right hand was studied in each subject except when this hand was deformed or injured. Table I records the dimensions of the three areas of the hand—in flexion, in extension and in the standard position.

Table II shows the average areas of the regions of the hand in three positions in the 10 subjects.

In Table III the area of each region of the hand is expressed as a percentage of the area of the control position. The average percentage of all 10 subjects is presented with the standard deviation. The maximum error with this method was 3.2%.

Observations on Technique

In developing this technique a number of technical and other difficulties had to be overcome. Many of the conclusions we reached through trial and error are incorporated in the following recommendations:

TABLE I.—Total Area of the Hand in Three Specified Positions

Subject no.	Age (years)	Height	Weight (lbs.)	Hand studied	Handedness	Area in flexion ($cm.^2$)	Area in extension ($cm.^2$)	Area in standard position ($cm.^2$)
1	23	5' 6″	134	right	left	341.079	340.710	339.108
2	44	6' 0″	181	left	right	514.170	495.117	499.381
3	53	5' 6″	112	right	right	— *	466.717	467.606
4	24	5' 3″	165	left	right	448.826	421.786	431.528
5	22	5' 5″	118	right	left	343.326	350.790	335.611
6	25	5' 6″	128	left	right	377.810	365.132	370.242
7	29	6' 2″	195	left	right	548.512	536.320	537.453
8	28	5' 8½″	150	right	left	386.423	377.444	383.222
9	21	5' 2½″	113	right	right	380.503	380.463	379.166
10	22	5' 9½″	150	left	right	469.570	458.322	471.432

*Subject number 3 refused to allow us to complete the moulding of the hand in flexion, i.e. his palm.

TABLE II.—Average Areas of the Regions of the Hand

Region	Area in flexion ($cm.^2$)	Area in extension ($cm.^2$)	Area in standard position ($cm.^2$)
Palmar			
Index finger	21.523 ± 3.148	24.488 ± 3.628	24.098 ± 3.868
Middle finger	23.195 ± 1.443	26.906 ± 4.724	26.556 ± 4.629
Ring finger	21.990 ± 4.357	24.666 ± 4.674	24.337 ± 4.413
Little finger	17.136 ± 3.656	18.798 ± 3.495	18.830 ± 3.559
Dorsal			
Index finger	31.281 ± 4.769	28.176 ± 4.709	28.098 ± 4.731
Middle finger	35.279 ± 5.196	31.900 ± 4.474	31.652 ± 4.621
Ring finger	30.890 ± 4.317	27.882 ± 4.702	27.975 ± 5.075
Little finger	21.476 ± 4.181	20.319 ± 4.619	20.168 ± 4.119
Palmar thumb	16.027 ± 4.515	16.588 ± 4.247	17.529 ± 4.394
Dorsal thumb	18.845 ± 2.280	18.072 ± 2.854	17.813 ± 2.159
Palm	62.472 ± 46.899	92.240 ± 47.712	78.908 ± 42.631
Dorsal hand	111.751 ± 17.070	90.414 ± 58.638	99.545 ± 45.118

TABLE III.—Area of Regions of the Hand as Percentage of the Standard (Mid) Position

Region	Flexion % of control	Extension % of control
Palmar		
Index finger	89.36 ± 3.91	101.52 ± 1.61
Middle finger	91.01 ± 3.29	101.33 ± 2.57
Ring finger	90.13 ± 2.55	101.28 ± 2.01
Little finger	91.09 ± 3.41	99.96 ± 2.75
Dorsal		
Index finger	111.68 ± 2.67	100.47 ± 2.42
Middle finger	111.59 ± 3.75	100.99 ± 3.16
Ring finger	113.28 ± 2.51	102.09 ± 3.64
Little finger	112.00 ± 4.03	101.08 ± 3.62
Palmar thumb	93.53 ± 5.35	97.50 ± 2.91
Dorsal thumb	105.72 ± 2.56	101.30 ± 4.32
Palm	89.36 ± 4.86	104.56 ± 1.90
Dorsal hand	112.15 ± 3.80	91.08 ± 4.70

The Silastic elastomer tends to polymerize spontaneously and therefore should be stored in a refrigerator.

The addition of the fluid, dimethylpolysiloxane, makes the plastic mould more flexible and hence easier to trim.

The Xerox copier tends to magnify the object photographed. To determine the degree of magnification, a graph of known dimensions was Xeroxed and the photocopy measured. All subsequent measurements had a deduction to allow for the percentage magnification. The figure should be under 2%; if it is above this value the copier should be adjusted.

The contrast between the black card and the plastic pieces also varies. Often the machine can be adjusted to give sharper outlines. If the lines remain indistinct they should be traced over with a sharp lead pencil to facilitate measuring.

In the Silastic mould the position of flexion was the most difficult to obtain because the digits stuck together. This difficulty was overcome by constructing individual moulds of the fingers and the palm. To obtain a good finger flexion mould we applied the catalyzed liquid to the palmar aspect of the finger in extension and then, before the Silastic polymerized, flexed the fingers and the wrist in that order. Similarly, the dorsum of the hand was coated with the catalyzed liquid and the hand extended before the plastic polymerized.

Pieces of unpolymerized plastic often adhere to the hand but are easily removed with xylol.

The technician and the patient should be warned that the lead catalyst reacts with the alloy in gold jewellery and may cause blistering of the skin. All such jewellery should be removed before moulding begins.

The accuracy of the measurements of an area increases with the number of times the prints are measured with a planimeter. Hence each print should be measured at least twice to eliminate error in readings of the planimeter scale and in calculating the number of units of area.

Folds or creases in the Silastic mould, which occur especially in flexion, are most easily printed by cutting the fold from the mould so that the sides are approximately equal and then photocopying one side. The true area is determined by multiplying the area of one side by two.

The subject must be constantly attentive if he is to maintain the specified position. This is especially true in extending the hand, because this position becomes uncomfortable long before the mould can be completed. Several patients complained of nausea, which they blamed on the smell of the plastic.

Discussion

As is obvious from Table I, the present study was not based on an adequate population sample; eight of the 10 subjects were in their third decade. To complete this project we should measure more hands in the age group 30 to 60 years. Subjects 3 and 4 could not be considered "normal"; subject 3 was approximately 20 lbs. underweight and 4 was 35 lbs. overweight.

Table II also shows the sampling error. The distribution of adult human hand areas is not "normal" according to the areas in the standard position. If one plots the area of the hand against the number of subjects whose measurements fall within the range of that area, there are two peaks and no one central tendency. This reflects the inadequate sampling, i.e. a larger number of subjects must be studied before we can draw conclusions.

In most of the subjects, as shown in Table II, the area is greatest in flexion and least in extension. In the standard position the area is somewhere between the two. Table III shows that the area of the dorsum of the hand changed 21% in moving from flexion to extension while the palm changed 15.20%. These data suggest that the dorsum of the hand stretches more on flexion than the palm does on extension and, therefore, that the total area in flexion is greater than that in extension. Al-

though this seems obvious when observing the hand in motion, it has never been demonstrated before. It is commonly stated that the dorsum of the hand expands 35% to 45% when the hand moves from the extended to the flexed position. This is one reason why the hand is maintained in the flexed position after a skin graft is applied to the dorsum. However, our results indicate that the expansion in flexion is considerably less than we had previously assumed.

The high standard deviations calculated by averaging the area of the various regions indicate that we studied a wide range of hands, and indeed this is necessary to obtain proper sampling. In contrast, the standard deviations derived from the percentage changes (Table III) had a maximum of 5%, which indicates a small range of changes in area from flexion to extension. With more studies these standard deviations may become highly significant.

It is evident from Table III that the areas of the dorsal and palmar aspects of the digits remain the same as the hand moves from the standard position to extension. This is because the digits cannot fully extend when the wrist is extended.

From flexion to extension the area of dorsal regions of the fingers changes more (12%) than the palmar regions (10%). Again, this indicates that the palmar aspect of the fingers does not stretch as much as the dorsal. The normal inability to hyperextend the joints, the thickness of the skin and the retinacular attachments to the skin also limit expansion of the palmar aspects of the fingers.

The average total area of the index finger is 52.8 sq. cm. This knowledge can be applied when using a flap from a filletted finger to resurface a skin defect of the adjacent dorsum of the hand. For example, if the index finger is useless, the phalanges can be removed and a vertical incision crossing the interdigital web to the level of the metacarpophalangeal joint will allow the finger flap to be advanced on to the dorsum of the hand. This flap can then cover a denuded area in excess of 50 sq. cm. Also, the distal end of this flap will extend to the level of the carpal bones. If palmar skin is divided horizontally at the metacarpophalangeal joint levels, thus releasing the tension of the flap while maintaining its blood supply through the neurovascular bundles, then the flap will extend further proximally. Skin defects on the distal segment of the thumb up to 2 cm. in vertical length can be repaired by advancing the skin of the anterior surface of the thumb (15.7 sq. cm.) distally; this can be done without producing a significant flexion deformity of the interphalangeal joint.

Summary

A method of calculating accurately the surface area of the hand and the digits has practical value in reconstructive surgery. The authors have described an original method of recording the surface markings of the hand and the digits. The surface areas are measured by means of a planimeter from Silastic moulds constructed with the hand in three different positions. In most of the subjects the area of the hand was greatest in the flexed position and least in the extended position. The area of the dorsum increased 21% in moving from extension to flexion while the palm area changed 15.2% in moving from flexion to extension. Although this seems obvious, it has not been demonstrated before by actual measurement. The surface area of the digits changes little in moving from the controlled position to the extended position. From the flexed position to the extended position, however, the change in digital area is almost 10%. This confirms the clinical observation that palmar skin does not stretch as much as dorsal skin.

The authors wish to thank Dr. J. O. Godden of Medi-Edit Limited, Toronto, for his advice during the preparation of this paper.

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Résumé

Pour pouvoir découper des lambeaux de peau en vue de greffes cutanées libres, il est évidemment nécessaire de mesurer avec précision la superficie de la main et des doigts. A cette fin, il faut naturellement commencer par définir les limites de ces régions, puisque les définitions existantes sont basées uniquement sur des repères anatomiques.

Chez 10 volontaires, on s'est servi d'un revêtement de Silastic pour construire des moules, les articulations étant en position anatomique, en flexion et en extension complète. Au moyen d'un duplicateur Xerox on a pris des "impressions" des moules dont la surface a été ensuite mesurée par un planimètre. Des corrections ont été apportées pour tenir compte des facteurs d'erreur.

Chez la majorité de ces sujets, la superficie de la main était plus grande en position fléchie et moindre en position d'extension. La superficie de la face dorsale a augmenté de 21% de l'extension à la flexion tandis que la face palmaire changeait de 15.2%. Sans doute, dira-t-on, ces faits semblent évidents à priori, mais ils n'avaient jamais été confirmés par une mesure précise. La région des doigts change peu de la position normale à la position d'extension, mais ce changement atteint tout de même près de 10% d'une position à l'autre. Ceci vient somme toute confirmer l'observation clinique que la peau de la paume ne s'étend pas autant que la peau de la face dorsale.

CHRONIC OR RECURRING ORGANOAXIAL ROTATION OF THE STOMACH*

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TORSION of the stomach about its longitudinal axis is called organoaxial rotation. Acute organoaxial volvulus is a well-known entity[1] and produces the triad of Borchardt:[2] intractable retching, severe epigastric pain, and inability to pass a Levin tube beyond the distal esophagus. Chronic or recurring organoaxial gastric rotation is more common than the acute form but its diagnosis may often be overlooked because it produces only vague or intermittent symptoms.

The stomach is suspended along its lesser curvature by the gastrohepatic ligament and along the greater curvature by the gastrocolic and gastrosplenic ligaments (Fig. 1). The gastrophrenic ligament is attached to the posterior aspect of the fundus[3] and attaches the stomach to the diaphragm. The stomach is held above by the esophagus; the peritoneum, binding the second part of the duodenum to the posterior abdominal wall, fixes the stomach below.

The factors that permit or promote unusual gastric mobility are: lax elongated gastric ligaments,[4,5] absent gastrocolic omental attachment,[6,7] the presence of duodenal mesentery, diaphragmatic defects, tumours of the stomach or of adjacent organs, inflammation and adhesions, ileus, and colonic distension. Radiologically, when a mercury-filled balloon is passed,[8] we have noted forward movement of the greater curvature during peristalsis even in normal stomachs.

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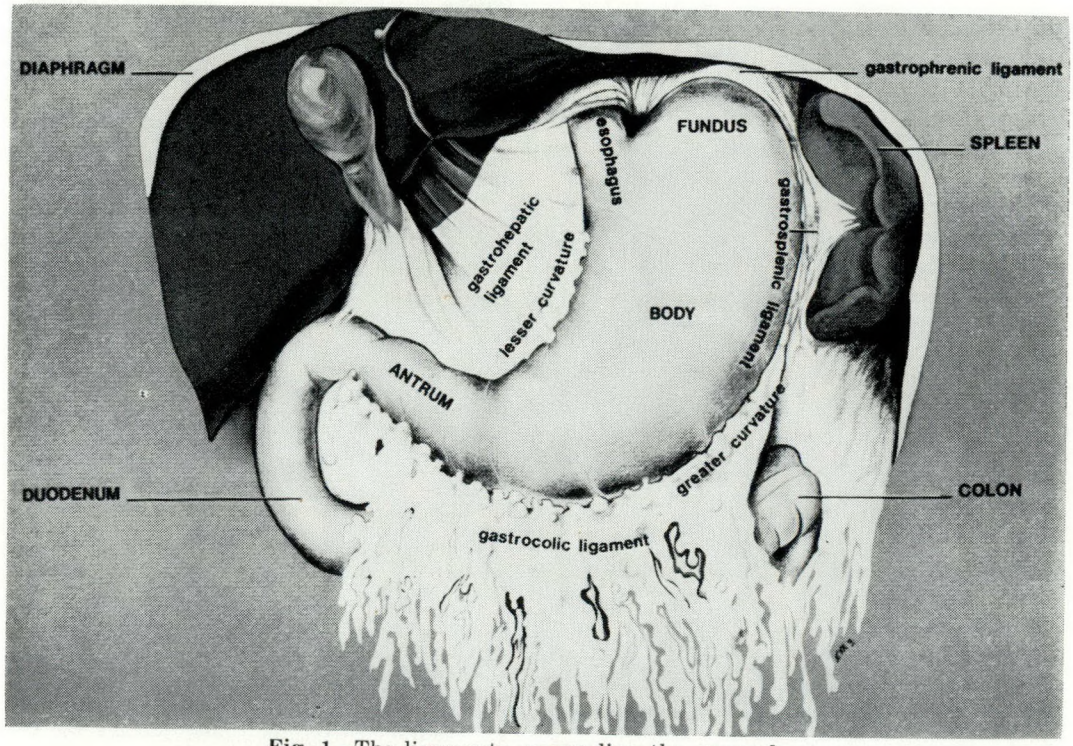
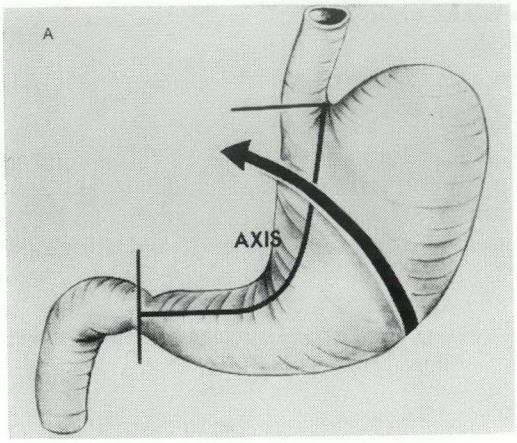
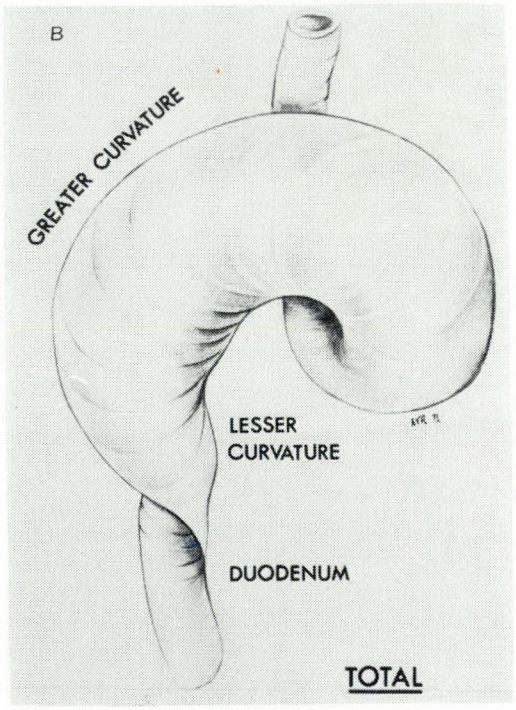
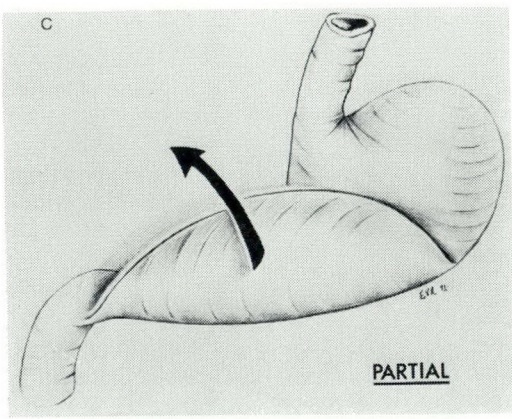


Fig. 1.—The ligaments suspending the stomach.







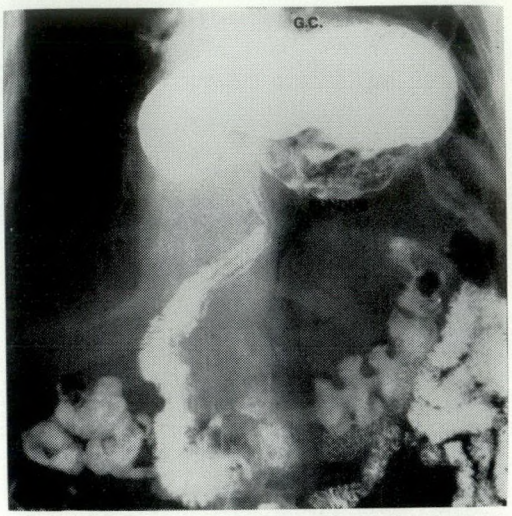


Fig. 3.—Patient no. 1. Intrathoracic stomach. The greater curvature (GC) lies uppermost. The ligaments have lengthened greatly.

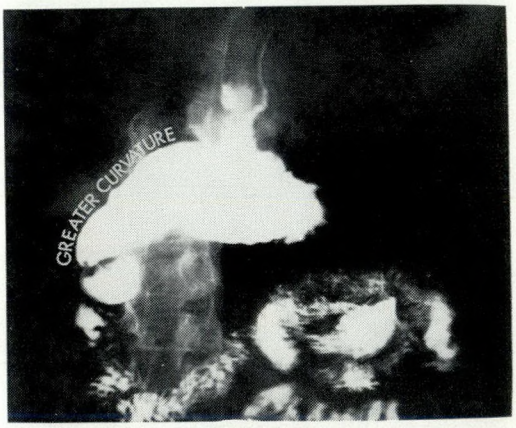


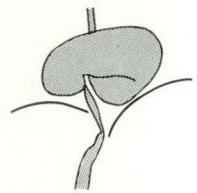
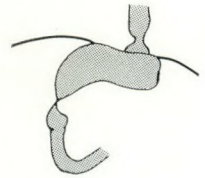
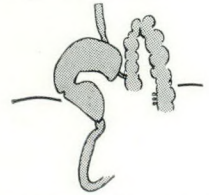
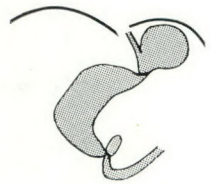
Fig. 4a

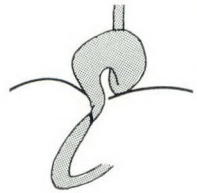
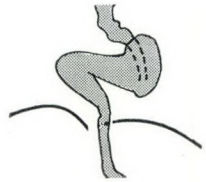
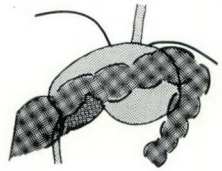
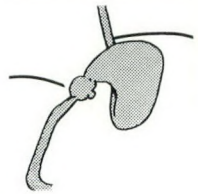
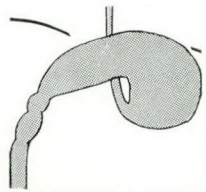
Fig. 4b

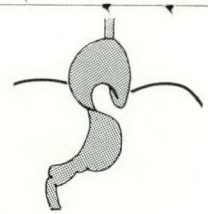
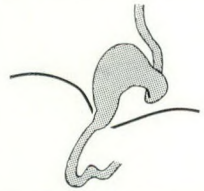
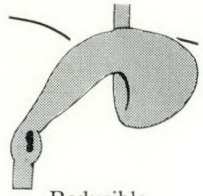
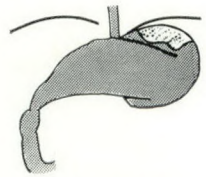
Fig. 4.—Patient no. 2. (a) The greater curvature lies uppermost against the diaphragm. (b) A bird-beak deformity (abrupt conical narrowing) at the site of the twist.

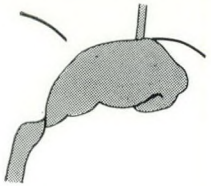
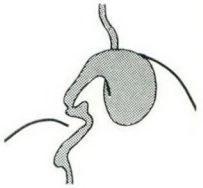
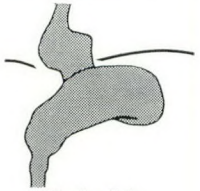
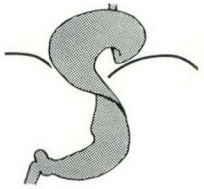
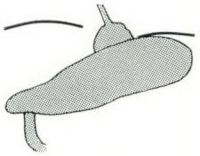
Fig. 2.—(A) The greater curvature rotates superiorly. (B) A twist at the irst part of the duodenum and the lower end of the esophagus (i.e. the entire stomach rotates). The pylorus may approach the cardia, folding the gastrohepatic ligament. (C) A twist occurring along the course (here the pars media) of the stomach (i.e. part of the stomach rotates).

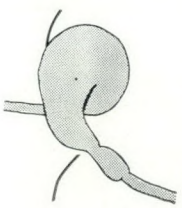
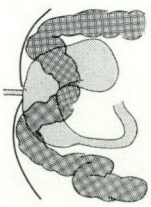
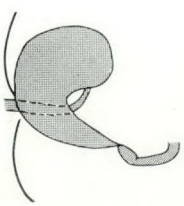
TABLE I.—CHRONIC OR RECURRING ORGANOAXIAL GASTRIC ROTATIONS ST. JOSEPH'S HOSPITAL, TORONTO, 1965-1971

Patient no.	*Sex*	*Age (years)*	*Year*	*Symptoms and signs of organoaxial rotation*	*Gastrointestinal series*	*Associated disease*	*Treatment*	*Follow-up*
1	F	68	1965	Single episode of upper abdominal pain lasting 24 hrs.		Hiatus hernia, intrathoracic stomach	Nasogastric suction	Lost to follow-up
2	M	79	1965	Upper abdominal distension and vomiting		Irreducible hiatus hernia, hemiplegic—old CVA, chronically constipated	Nasogastric suction, then bland diet	Mild epigastric discomfort, further cerebral thrombosis
3	M	88	1966	Epigastric burning, fullness, coffee-ground vomitus		Huge hiatus hernia —¾ of stomach incarcerated above diaphragm, fistula between proximal stomach and herniated transverse colon, hemiplegic	None, prohibitive operative risk	Readmitted 4 months later with coffee-ground emesis, died
4	F	52	1966	3 attacks of pain, following distal pancreatectomy and splenectomy for carcinoma tail of pancreas, considerable delay in gastric emptying (volvulus created surgically)			None—no further attacks, volvulus persisted, but no delay in emptying on 1967 GI series	Died 1968 of metastases from carcinoma of pancreas

5	F	74	1967	Epigastric fullness		Substernal burning after meals or on bending, hiatus hernia with reflux	Repair of hiatus hernia (through an abdominal approach) and gastrostomy	Asymptomatic
6	F	76	1967	Infrequent retching and upper abdominal pain		Intrathoracic stomach	None	Same
7	M	48	1967	None		Cholecystitis	Cholecystectomy and anterior gastropexy	Asymptomatic
8	M	50	1967	None		Mid-epigastric burning from duodenal ulcer, eventration of left diaphragm	Bland diet — ulcer healed	Died of carcinoma of lung, 1969
9	F	62	1967	Heartburn and epigastric pain, "gas"			Antacid	Asymptomatic

10	F	70	1968	Intermittent nausea, vomiting immediately after meals		Hiatus hernia with considerable reflux, severe rheumatoid arthritis (steroids), arteriosclerosis	Bland diet	Died of myocardial infarction 10 months later
11	F	81	1969	Tarry stools, some mid-epigastric discomfort before meals		Intrathoracic stomach	Bland diet and antacid	Died 1971, ruptured aortic aneurysm
12	M	60	1969	None	 Reducible	Severe mid-epigastric burning, duodenal ulcer	Bland diet —ulcer healed	Asymptomatic
13	F	78	1969	None		Obstructive jaundice — choledocholithiasis, large hiatus hernia with reflux	Cholecystectomy, common duct exploration, and anterior (Nissen) gastropexy	Asymptomatic
14	M	88	1969	None	 Reducible, transverse stomach	GI hemorrhage from leiomyosarcoma of fundus	Resection of tumour of fundus	Died 1971, CVA

15	F	66	1969	None		Cholecystitis	Cholecystectomy	Asymptomatic
16	F	79	1969	None (GI series done because of air bubble on chest film)		Eventration left leaf of diaphragm	None	Asymptomatic
17	M	65	1969	Single attack—severe mid - epigastric pain and vomiting	 Reducible	Hiatus hernia	None	Asymptomatic
18	F	88	1970	None		GI hemorrhage from large prepyloric ulcer. Large hiatus hernia, arteriosclerosis, diabetes, osteoarthritis	Bland diet—ulcer healed	Asymptomatic
19	F	60	1970	Epigastric distress and vomiting	 Reducible, transverse stomach	Small hiatus hernia with reflux, severe osteoarthritis, angina pectoris	Bland diet—discontinue aspirin	Improved

20	F	73	1971	None	Reducible 	Cholecystitis, eventration of left diaphragm	Cholecystectomy	Asymptomatic
21	M	62	1971	Upper abdominal and retrosternal pain and fullness, belching	Infracolic 		Bland diet and antacid	Asymptomatic
22	M	36	1971	3 years episodic mid-epigastric and low retrosternal pain and discomfort, delayed gastric emptying		Marked obesity	Refused operation, bland diet, weight reduction	

Organoaxial torsion occurs about the long (cardiopyloric) axis of the stomach (Fig. 2a). The greater curvature rolls anteriorly (rarely posteriorly) and comes to lie superior to the lesser curvature. The rotation may be total if the whole stomach rotates (Fig. 2b) or partial if only a segment of the stomach rotates (Fig. 2c). The transverse colon may be elevated but the stomach usually remains supracolic, although infrequently it may assume an infracolic position.[2, 6, 9, 10]

In acute complete (i.e. $> 180°$) organoaxial volvulus[11] the pylorus and cardia close off, producing a closed loop with the onset of dramatic pain, epigastric distension and neurogenic shock. If a tube cannot be passed gently into the stomach, emergency surgical reduction is necessary.[9, 12] The chronic, incomplete or recurring organoaxial rotation may be accompanied by epigastric discomfort or hematemesis because of delayed gastric emptying and stasis, brief episodes of complete obstruction, or no symptoms.

Since 1965, chronic organoaxial rotation has been demonstrated radiologically in 22 patients at St. Joseph's Hospital, Toronto, Ontario. Occasionally, an organoaxial rotation may be produced transiently during barium study of the stomach, by the position adopted or by pressure-displacement, but generally organoaxial rotation was consistent in these 22 pa-

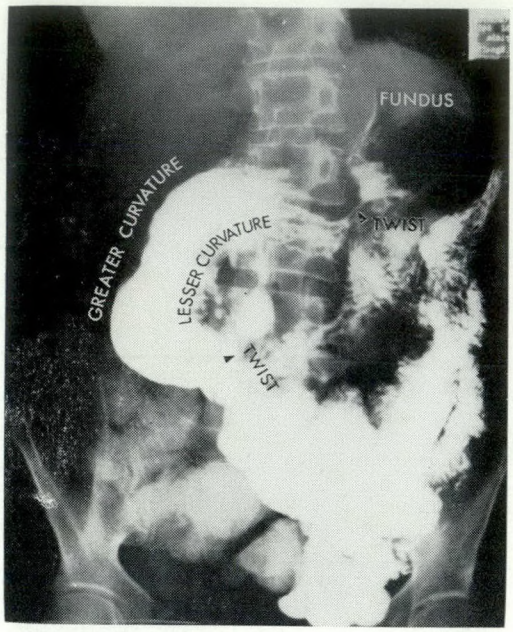


Fig. 5.—Patient no. 4. Organoaxial rotation of the distal stomach with a delay in emptying. The twist (beak) has occurred along the course of the stomach and at the pylorus. (Here, partial organoaxial rotation was produced by an operative procedure in which the gastrocolic ligament had been incised and the stomach rotated.)

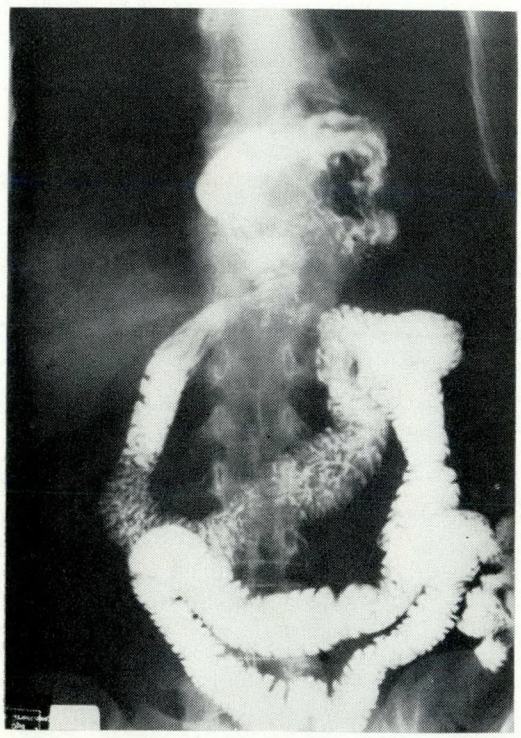


Fig. 6a

tients. The configurations and clinical features are set forth in Table I. Nine of these patients had no symptoms. Many had postprandial epigastric pain, bloating and eructations which improved on a bland diet. Of those who had symptoms, five were elderly and were poor surgical risks because of cardiac disease, hemiparesis, or debilitating arthritis. They were treated con-

Fig. 6b

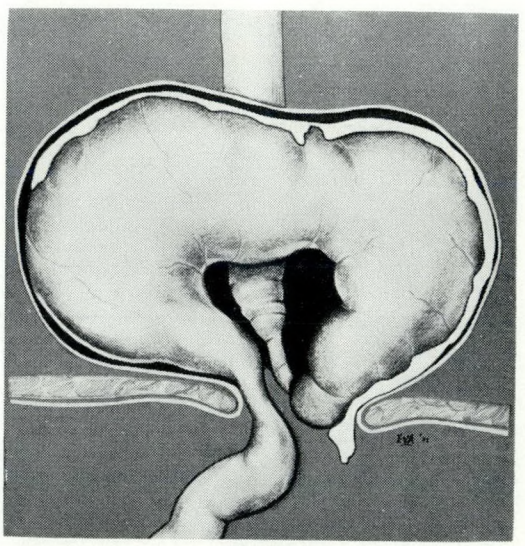


Fig. 6c

Fig. 6.—Patient no. 5. (a) "Upside-down" stomach in hiatus hernia. (b) Close-up of (a). (c) A diagram of (b). Note that, in organoaxial rotation, what is normally the posterior wall of the stomach faces anteriorly and is covered by the gastrocolic omentum which is attached to the greater curvature.

Fig. 7a

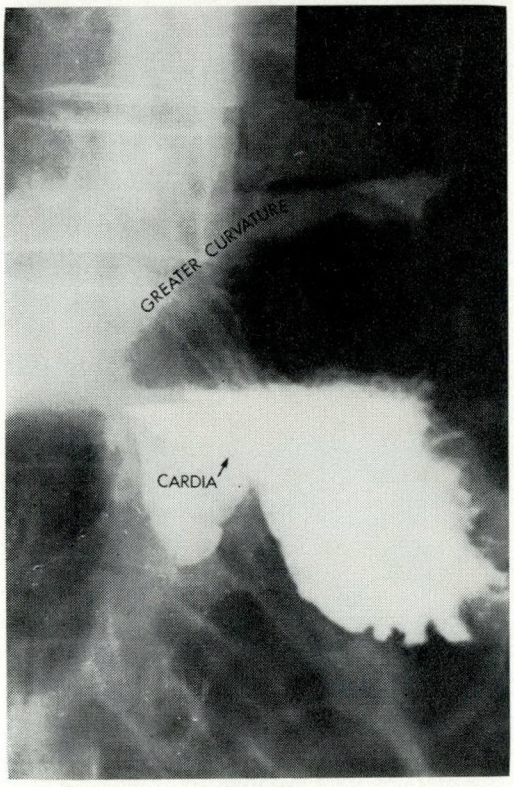


Fig. 7b

Fig. 7.—Patient no. 6. (a) The transverse colon rises with the stomach (see Table I). (b) The esophagus enters the dependent portion of the stomach (lesser curvature). (See also Fig. 9.) This differs from a "cascade" stomach, where the fundus falls posteriorly but the esophagus enters superiorly.

Fig. 10.—Patient no. 12. Organoaxial gastric rotation showing a duodenal ulcer which healed with a bland diet.

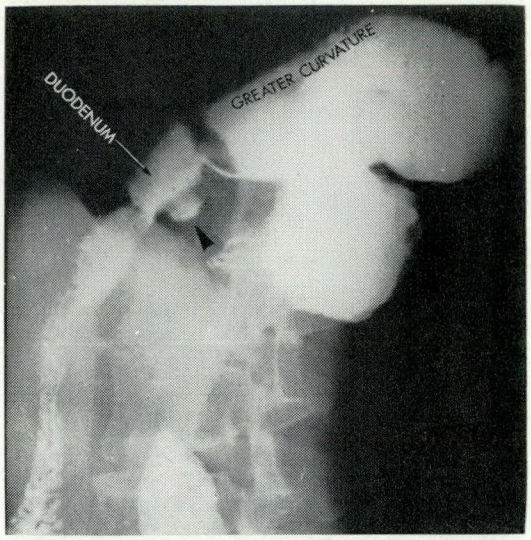


Fig. 8.—Patient no. 12. Eventration of the left diaphragm with an "upside-down" stomach. The arrow points to an ulcer in a highly mobile duodenum; this ulcer shows the excavation often seen with gastric ulcers rather than the usual crater seen in a duodenum bound by inflammation and scar. The ulcer healed with a bland diet but the rotation of the stomach persisted.

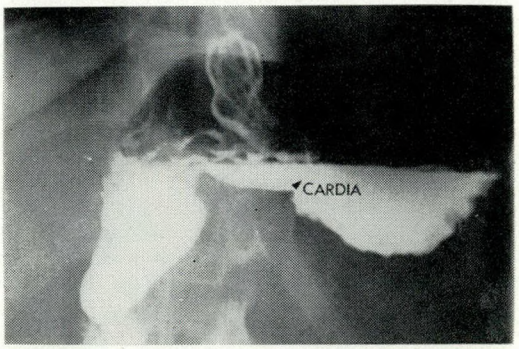


Fig. 9.—Patient no. 9. The greater curvature lies uppermost, adjacent to the diaphragm.

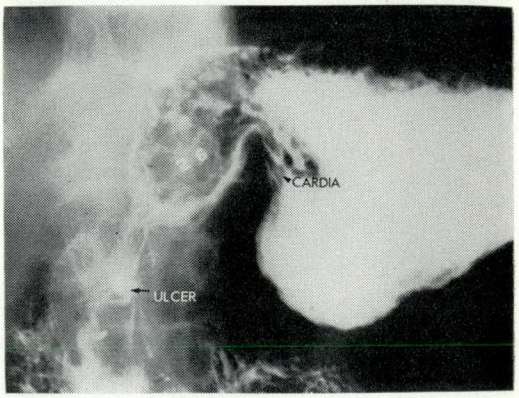


Fig. 11.—Patient no. 15. Organoaxial volvulus of the stomach.

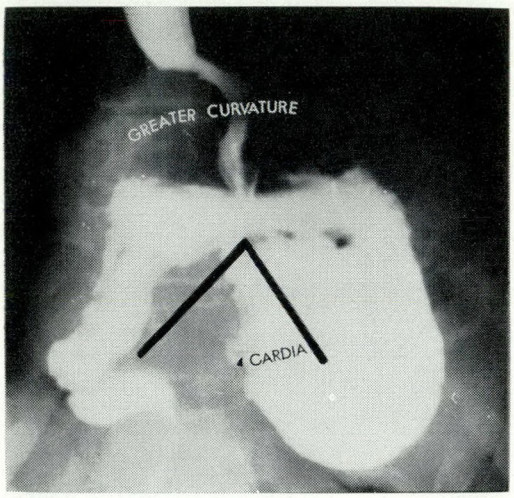


Fig. 12.—Patient no. 16. Eventration of the left diaphragm. The pylorus approaches the cardia and an inverted V occurs in the lesser curvature (see also Figs. 2b and 7b).

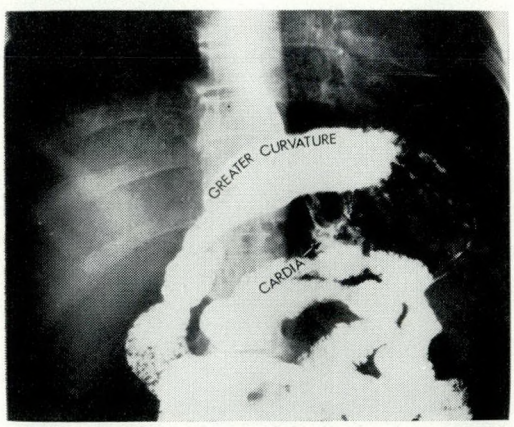


Fig. 13.—Patient no. 21. Dependent cardia. Note that the rotated greater curvature forms a convexity continuous with the duodenum.

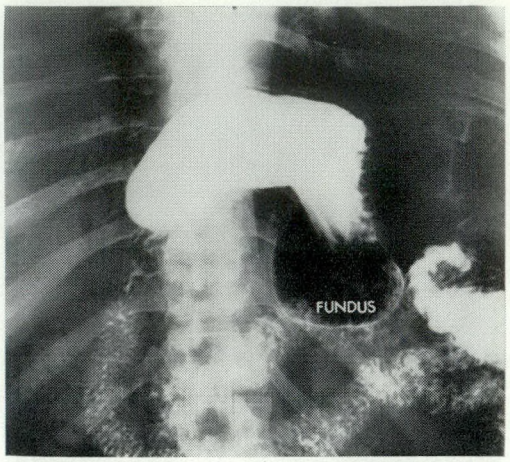


Fig. 14.—Patient no. 21. Following this study, a barium enema (combined with some barium by mouth) showed this rotation to be essentially infracolic (see Table I).

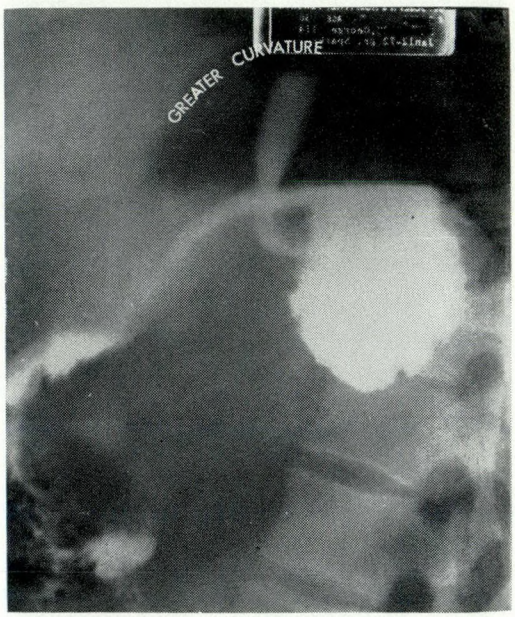


Fig. 15.—Patient no. 22. A good example of an "upside-down" stomach.

servatively and generally died of their medical diseases. Figs. 3 to 15 illustrate upper gastrointestinal barium radiographs of these stomachs (compare with Table I).

If symptoms persist or recur and are severe, operation is necessary. After "detorsion" the mobile stomach is anchored by one or a combination of the

following: (1) anterior gastropexy (tethering the anterior gastric wall securely to the anterior abdominal wall),[5, 7, 13] (2) gastrostomy alone or with (1),[7] (3) gastroenterostomy or Billroth I gastrectomy, (4) correction of the diaphragmatic defect when possible, (5) suture of the gastrocolic ligament to anterior parietes,[14] or plication of the gastric ligaments to reduce their width.

Conclusion

Chronic or recurring organoaxial gastric torsion produces symptoms of delayed gastric emptying, brief episodes of obstruction, or no symptoms. Although the surgical emergency, acute organoaxial volvulus, has been widely reported, chronic rotations should not be overlooked as a possible symptom-producing entity.

The author wishes to acknowledge the assistance of Drs. Wallace Roy, I. Y. Nagarwalla, M. Tam, J. A. McIntyre, A. Haber, M. Baida, E. Grundy and L. Mautner, Mrs. M. Shadd, and the Department of Photography, St. Joseph's Hospital, Toronto.

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Résumé

On appelle rotation organo-axiale de l'estomac la torsion de l'estomac autour de son grand axe, de sorte que la grande courbure se trouve située le plus en dessus. Le volvulus organo-axial complet aigu est une urgence classique. La rotation organo-axiale chronique ou récidivante est une découverte plus courante qui peut être méconnue parce qu'elle ne donne que des symptômes assez vagues ou intermittents, par suite d'un retard de la vidange gastrique ou à l'occasion de brefs épisodes d'obstruction.

L'auteur rapporte les cas de 22 malades qui présentaient une rotation organo-axiale chronique, d'après l'image radiologique généralement obtenue. Neuf d'entre eux étaient asymptomatiques. Plusieurs autres avaient une douleur épigastrique postprandiale, du gonflement et des éructations, symptômes qu'améliorait un régime d'épargne gastrique. Parmi les malades symptomatiques, on comptait cinq malades âgés qui, étant de mauvais risques chirurgicaux reçurent un traitement conservateur. Ils sont généralement décédés de maladies médicales.

Parmi les facteurs qui ont favorisé la torsion de l'estomac, figuraient une élongation des ligaments gastriques, un mésentère duodénal, des anomalies diaphragmatiques, une tumeur de l'estomac ou d'organes adjacents, un iléus et une distension du côlon. Dans les cas où les symptômes persistaient ou récidivaient et étaient sévères, il a fallu opérer. Après avoir corrigé la torsion, l'estomac mobile a été ancré par une ou plusieurs des méthodes suivantes: (1) gastropexie antérieure, (2) gastrostomie, (3) gastroentérostomie ou gastrectomie Billroth I, (4) correction de l'anomalie diaphragmatique, (5) plicature des ligaments gastriques.

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VIABILITY OF SKIN GRAFTS STORED IN VARIOUS MEDIA*

L. N. HURST, M.D., F.R.C.S.[C], W. K. LINDSAY, M.D., M.S., F.R.C.S.[C], F.A.C.S. and J. LEE, B.Sc., *Toronto, Ont.*

THE use of preserved skin for transplantation has become commonplace. Excess skin taken at the original grafting operation can be stored and applied later without the necessity of a further general anesthetic. The uses of such grafts range from resurfacing small areas to improve a clinical result and shorten hospitalization, to providing temporary biological dressings for major burns. Such dressings may be lifesaving.[1]

Skin can be preserved by freeze-drying or lyophilization and storage at room temperature, by freezing and storing at temperatures below 0° C., or by refrigeration at 4° C. The first two methods require expensive equipment so that to be practical the demand must be high enough to justify such facilities. Such demand is rare outside specialized institutions and therefore refrigeration is the commonest method used today.

In 1896 Wentscher[2] first reported the clinical use of skin preserved by cooling. Since then this type of tissue preservation has been improved.[3-7] The method appears deceptively simple, but results may be disappointing if the storage temperature is improperly controlled, the storage medium inappropriate or the storage time too long.

Ideally the skin should be stored at a contant temperature of 4° C.[8] Above this, metabolism in the graft increases and consequently the period of viability decreases. Temperatures below 4° C. favour ice-crystal formation in the graft resulting in hyperosmolar damage. Oxygen utilization decreases with cooling but enough must be present to support cellular metabolism, so the storage container must contain air. The medium in which the skin is stored should supply electrolytes and nutrients in physiologic concentration, and be buffered against the acid metabolites constantly produced by the graft.

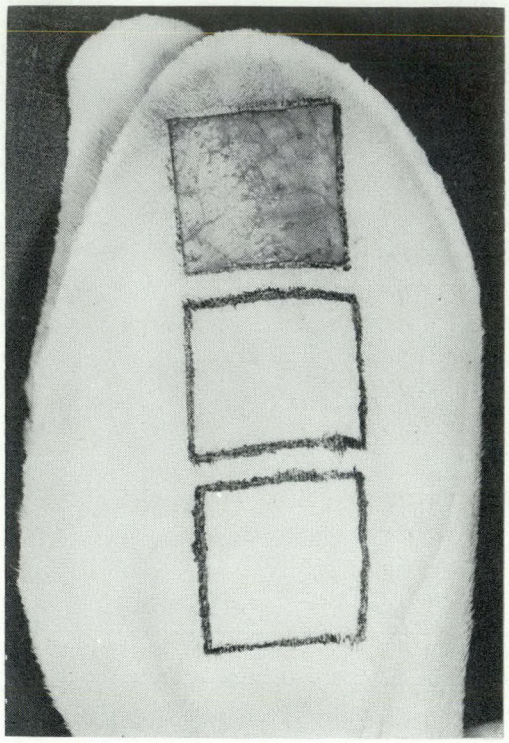

Fig. 1.—Rabbit's ear marked out in squares: a full-thickness skin defect has been created in the distal square.

This paper reports a study designed to evaluate and to improve the efficiency of the skin storage facility at The Hospital for Sick Children, Toronto.

METHOD

We used 30 male albino rabbits as experimental animals because their ears provide accessible grafting sites. Three squares, 2 x 2 cm., were outlined with India ink on each ear (Fig. 1). Scrotal skin was removed, cut into six squares of like size, and placed on gauze in Petri dishes containing one of three storage solutions: Ringer's solution, Earle's solution, and Earle's solution with 10% by volume of rabbit serum added. Earle's solution is a tissue culture medium containing sodium, potassium, calcium, magnesium, phos-

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TABLE I.—EARLE'S SOLUTION

NaCl	6.8 g.
KCl	0.4 g.
$CaCl_2$	0.2 g.
$MgCl_2$	0.2 g.
$NaHPO_4$	0.125 g.
$NaHCO_3$	2.2 g.
Dextrose	1.0 g.
Water	1000 ml.

phate, 1 g. of dextrose and 2.2 g. of sodium bicarbonate per litre (Table I). The pH ranges from 7.2 to 7.4. We added gentamicin sulfate 20 mg./100 ml. to all three storage solutions and provided 2.4 ml. of solution for each square centimetre of skin stored. The skin and solution were placed in Petri dishes which allowed an air space between the surface of the medium and the lid. The Petri dishes were sealed and stored in a refrigerator at 4° C.

Using two rabbits as test animals for each solution, we determined the approximate life span of grafts stored in each solution. The experimental protocol bracketed this period of viability. Thus, storage time differed with the solution used, but the first graft from each was placed on day one to establish a base line. Subsequently, at the predetermined period of time after the initial operation, a full-thickness defect down to perichondrium was created in the rabbit's ear, starting distally. The stored skin was applied, sutured in place and covered by a bolus dressing which was removed 10 days later and the initial "take" of the graft assessed. A second assessment was made at 20 days (Fig. 2).

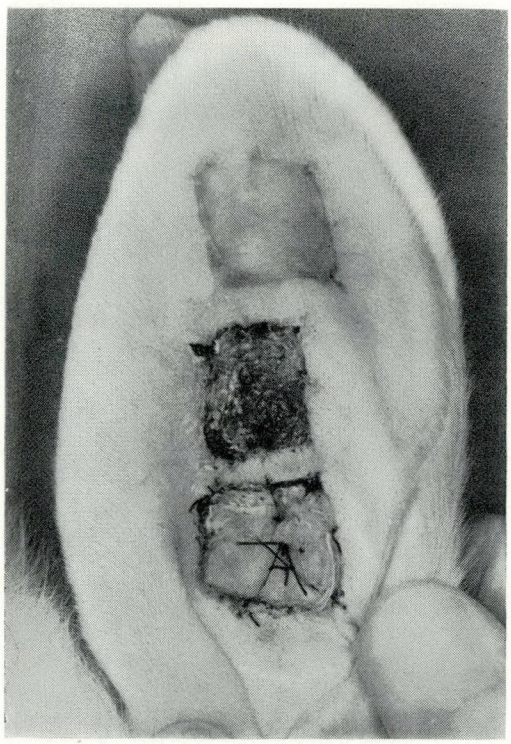

Fig. 2.—Distal square shows complete take of a graft stored for five days. Middle square shows failure of a graft stored for 10 days. A bolus dressing is still in place over the proximal graft.

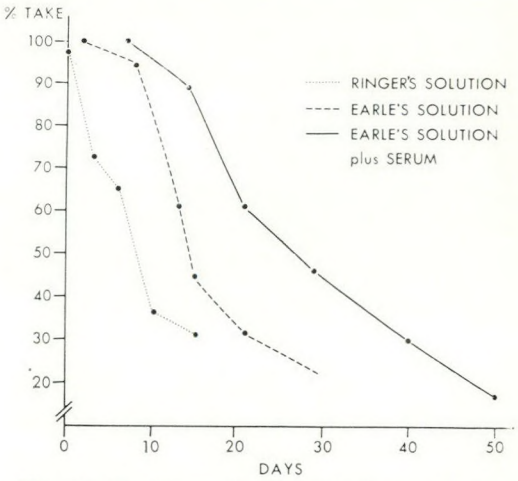

Fig. 3.—Graft survival related to storage time in different solutions.

RESULTS

The results are graphically shown in Fig. 3.

Initially all grafts stored in Ringer's solution "took" completely. However, viability fell rapidly to less than 50% at the end of seven days' storage as measured by the amount of initial take. Grafts stored in Earle's solution showed an extended viability. Takes of 100% were achieved after up to eight days' storage, then declined. The average graft produced a 50% or better take at the end of 13 days' storage as compared with seven days for Ringer's solution. Storage in Earle's solution plus serum again extended the viability of the pre-

served grafts. Takes of 100% were observed after more than two weeks' storage with a gradual decline after this. The average graft showed a 50% or better take at 22 days. The effect of altering the storage medium to a more physiologic solution progressively extends the usable storage period as represented by a marked shift from Ringer's solution on the left to Earle's solution plus serum on the right.

The quality of the grafts varied considerably depending on the solution in which they were stored. Originally the grafts were strong, pliable and elastic. When stored in Ringer's solution for five days, the graft was markedly edematous, unduly friable and completely inelastic. When stored in Earle's solution alone or Earle's solution plus serum for up to 50 days the graft showed little change. It was neither edematous nor unduly friable and retained its elastic properties.

Discussion

A skin graft is a sample population of cells of differing ages and degrees of vitality. Factors preserving the viability of these cells will increase the chances of graft take. For practical purposes the half-life of the graft, that amount of time after which the average take is 50%, may be considered the endpoint of useful storage. As shown in Fig. 4 the graft half-life stored in Ringer's solution is seven days, in Earle's solution it is 13 days and in Earle's solution plus serum it is 22 days.

If, during an operative procedure, excess skin is taken and preserved, it must be stored for more than seven days because more time is required to assess the original take and, if the latter is inadequate, to prepare a suitable bed for regrafting. It is futile to apply preserved skin over a sloughing bed. This time factor makes Ringer's solution an unsatisfactory storage medium.

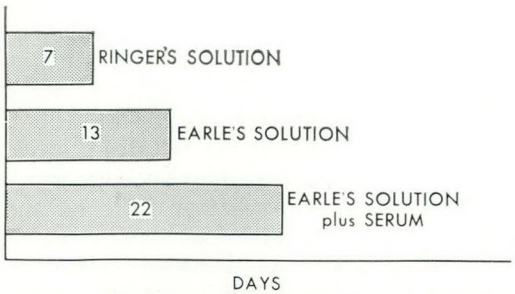

Fig. 4.—Useful storage time in various solutions.

Earle's solution is better and extends the useful storage time to 13 days. Earle's solution plus serum increases the time still further to 22 days, a period of practical clinical significance because, by then, adequate time has elapsed for the graft to be used to the best advantage.

Earle's solution comes in 400-ml. bottles and is easily obtainable through normal hospital suppliers. Ten per cent pooled serum obtained through the Red Cross and antibiotics should be added. We use 80 mg. of gentamicin sulfate for each bottle of storage medium. Cultures taken at the time of grafting have all been sterile. The completed solution can be stored in a refrigerator at 4° C. for up to six months without deteriorating. Consequently it meets the first criterion for use in any hospital—that of convenience. Once the supply and procedure for preparation have been established the solution may be used as readily as saline and viable skin may then be stored for longer periods of time.

The results of animal experiments must always be applied cautiously to the treatment of patients. However, as a general guideline, the commonly used intravenous solutions should be replaced by a more physiologic tissue-culture medium for the storage of skin grafts.

Summary

The methods of skin preservation have been reviewed with emphasis on the commonly used refrigeration method. Rabbit skin grafts were employed to demonstrate the superiority of a nutrient tissue-culture medium over the more frequently used intravenous solutions. Its clinical application has proved to be convenient and the results have proved much more successful than those from previous methods of storage.

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Résumé

Les auteurs passent en revue les diverses méthodes de conservation de la peau et insistent particulièrement sur la méthode classique de la réfrigération. Ils ont utilisé des greffes cutanées de lapins pour démontrer la supériorité du milieu de culture tissulaire nutritive sur les solutés intraveineux couramment utilisés. Son application clinique s'est révélée commode et ses résultats ont amplement démontré que cette méthode de conservation réussit mieux que les méthodes antérieures.

RELATIONSHIP BETWEEN VAGOTOMY, PEPTIC ULCER AND GASTRIC ADENOCARCINOMA IN RATS FED 2,7-DIACETYLAMINOFLUORENE*

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THE purpose of the present study was to determine whether post-vagotomy gastric achlorhydria and/or the presence of healing peptic ulcer in rats influence the morphology of gastric mucosa exposed to the oral carcinogen 2,7-diacetylaminofluorene (2,7-DAAF).

Achlorhydria is frequently associated with gastric carcinoma, but the etiologic correlation between the two conditions remains obscure. These two conditions appear to be uniquely human diseases. We were able to produce achlorhydria experimentally in various laboratory rodents by prolonged parenteral administration of an anticholinergic drug.[1, 2] We also found that achlorhydria enhanced the malignant transformation of gastric mucosa in rats with a carcinogen implanted into the stomach wall.[3] Achlorhydria can be easily produced in rats by truncal vagotomy.[4] In these animals, interdigestive gastric secretion or "spontaneous" gastric secretion is identical with the cephalic phase[5, 6] and can be completely suppressed by vagotomy.[4, 5] Vagotomy has been used previously in an attempt to enhance gastric carcinogenesis in rats. Morgenstern[7] found that gastrojejunostomy alone or combined with vagotomy enhanced the carcinogenic effect of methylcholanthrene (MCH). In his study, suture threads in the gastric wall were saturated with the carcinogen, MCH, and most lesions developed in the vicinity of the anastomoses.

Some clinicians[8-11] consider that peptic ulcer and gastric tumours have an etiologic relationship but this remains a matter of controversy. We found recently that the presence of a healing peptic ulcer, experimentally induced, enhanced the appearance of gastric adenocarcinoma in rats fed the carcinogen, 2,7-DAAF.[12]

Gastric adenocarcinoma is difficult to produce[13, 14] in laboratory animals. It was accomplished experimentally in a very small percentage of animals by direct implantation of a potent carcinogen into the gastric wall,[13, 15] or by prolonged administration of a carcinogen *per os* or parenterally.[12, 16, 17] From a clinical point of view, gastric tumour produced by local implantation of a carcinogen differs from one induced by a carcinogen present in the food. Exposure to a carcinogenic agent in food may be one of the causes of gastric cancer in human subjects.

We concluded that vagotomy is the simplest method of inducing achlorhydria in rats, and that feeding a carcinogen is the best "clinical approach" to experimental gastric malignancy. We therefore decided to use an oral carcinogen in vagotomised rats. In some rats we also combined vagotomy with experimental gastric ulcer and then fed them the carcinogen. By these methods we set out to evaluate the effect of vagotomy and of the healing of peptic ulcer on the carcinogenic action of oral 2,7-DAAF in rats.

METHODS

Male Buffalo strain rats used for this study were obtained from Microbiological Association Inc., Walkersville, Md.

Surgery

All surgical procedures were performed under Diabutal anesthesia—44 mg./kg. body weight. After gastrotomy, penetrating gastric ulceration, measuring about 6 mm. in diameter, was produced by electrothermocautery[18, 19] in the mid-portion of the posterior wall of the glandular stomach proximal to the pylorus. Vagotomy was carried out on both nerve trunks using Lambert's technique.[20] When both operations were performed in the same animal, they were completed on the same occasion.

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Diet

Standard Purina food was the basic diet. Carcinogen diet consisted of basic diet with 0.25 g. of the carcinogen (2,7-DAAF) added to 1 kg. of dry diet.[12] It has been shown previously that this compound is selectively deposited in the gastric mucosa of the rat.[21]

Design of the Experiment

We used the following groups of animals: (1) no operation, fed basic diet; (2) peptic ulcer and basic diet; (3) vagotomised, fed basic diet; (4) vagotomised, peptic ulcer and basic diet; (5) no operation, fed 2,7-DAAF; (6) peptic ulcer and 2,7-DAAF; (7) vagotomised, 2,7-DAAF; (8) vagotomised, peptic ulcer and 2,7-DAAF.

Rats were kept on basic or carcinogenic diet for several months and observed daily for their general health. Body weight and food consumption were measured twice weekly. The animals were sacrificed after 46 weeks. Rats which had a palpable abdominal mass, or in which death appeared imminent, were sacrificed earlier. Autopsy was performed on all animals. Histologic studies, limited to gastric tissue, were done after staining with hematoxylin and eosin.

RESULTS

Table I summarizes some striking histologic findings in stomachs of rats exposed to an oral carcinogen. This table does not include histology of experimentally produced peptic ulcer. This subject was discussed in our recent report,[12] which also commented upon typical hepatic lesions following the administration of 2,7-DAAF to rats. The present study was concerned with malignant lesions of glandular mucosa. We refer, however, to some other abnormalities of gastric mucosa which may or may not be related to both the operation and the carcinogenic diet.

The terminology used in Table I generally conforms to the detailed description given in a previous report[3] published in this journal. Thus, gastric mucosal atrophy was indicated by generalized thinning of the glandular mucosa associated with atrophy or loss of zymogenic granules, chief cells and/or parietal cells; however, we did not measure the thickness of the mucosa. Intestinal metaplasia was considered present when there was unequivocal evidence of transformation of the gastric mucosa from a normal pattern of glands and cells to a type consistent with intestinal mucosa.[3]

Some of the lesions enumerated in Table I may be considered pre-cancerous.[3] We will not discuss this controversial problem[3, 12, 15] because our major purpose was to determine the incidence of gastric adenocarcinoma in animals fed carcinogen. A total of 28 such carcinomas were found in 148 stomachs of rats exposed to 2,7-DAAF, and no tumour was present in controls given no carcinogen. The general histologic char-

TABLE I.—DETAILED CLASSIFICATION OF SOME ABNORMALITIES OF GASTRIC MUCOSA FOUND IN FOUR GROUPS OF RATS FED A DIET CONTAINING THE CARCINOGEN 2,7-DIACETYLAMINOFLUORENE

	Rats exposed to oral carcinogen			
Histology of gastric mucosa	*No operation*	*Gastric ulcer*	*Vagotomy*	*Vagotomy + gastric ulcer*
No. of stomachs	16	40	43	49
Atrophy	2	2	20	11
Intestinal metaplasia	1	1	3	3
Glandular dilatation	2	7	4	3
Atypical glands, no penetration	—	1	1	1
Epidermoid carcinoma*	2	—	1	1
Adenocarcinoma	—	6	6	8
Anaplastic adenocarcinoma	—	1	—	1
Mixed adeno- and epidermoid-cell carcinoma†	1	3	2	—

*These tumours were localized in the gastric rumen.
†These tumours were localized at the junction between glandular mucosa and rumen.

TABLE II.—Effect of a Carcinogen 2,7-Diacetylaminofluorene, Ingested in Food, on Gastric Pathology in Rats

Group	No. of rats	Average body weight grams		Gastric ulcer	Vagotomy	Carcinogen	*Duration of observation in weeks	Gastric tumour		
		Initial	Final					†Adeno-carcinoma (Ad)	Ad + Ep	Epidermoid carcinoma (Ep)
1	16	140	338	−	−	−	45	−	−	−
2	20	148	340	+	−	−	40 — 45	−	−	−
3	20	138	280	−	+	−	38 — 46	−	−	−
4	20	146	226	+	+	−	30 — 46	−	−	−
5	16	140	230	−	−	+	26 — 46	−	1(6)	2(12)
6	40	122	211	+	−	+	26 — 46	7(17)‡	3(8)	−
7	43	148	251	−	+	+	12 — 46	6(14)	2(4)	1(2)
8	49	155	209	+	+	+	26 — 46	9(18)	−	1(2)

*This also corresponds to the duration of feeding carcinogen to groups 5 to 8.
†Earliest adenocarcinoma was found in a rat of group 6, after 32 weeks of exposure to 2,7-DAAF.
‡Values in brackets express percentage (%) of rats with tumours, in a given group.

acteristics of these adenocarcinomas agree with descriptions previously reported[3, 12, 15] and we will not discuss them in detail. Tumours are classified as purely glandular (22 adenocarcinomas) and "mixed"—containing both glandular and epidermoid tumour cells (six tumours). A neoplasm was considered to be adenocarcinoma if it showed glandular epithelial tumour elements penetrating all muscular coats of gastric wall.[3] In most instances the invasion extended to the serosa.[15] In groups 7 and 8 we did not always demonstrate that the malignant lesion localized in the former scar of peptic ulcer. In some instances infiltration of the wall by tumour did not permit identification of the ulcer scar.

Table II shows that group 5, those with no operation, had only one malignant mixed tumour. In the remaining three groups, adenocarcinomas and mixed tumours were present in 18% to 25% of rats. This observation suggests an etiologic relationship between peptic ulcer and/or vagotomy and malignant transformation of gastric mucosa in 2,7-DAAF-fed rats. Some of the tumours are illustrated in Figs. 1 to 8.

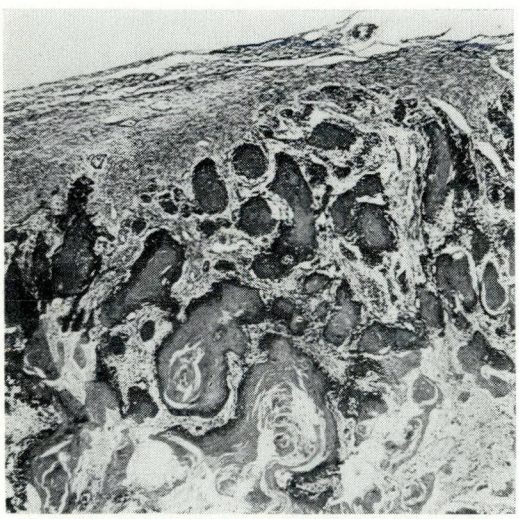

Fig. 1.—Low-grade, gastric, epidermoid-cell carcinoma in a rat that had no operation and was fed carcinogenic diet for 27 weeks. This figure demonstrates epithelial masses in a loose fibrous stoma (H & E x 25).

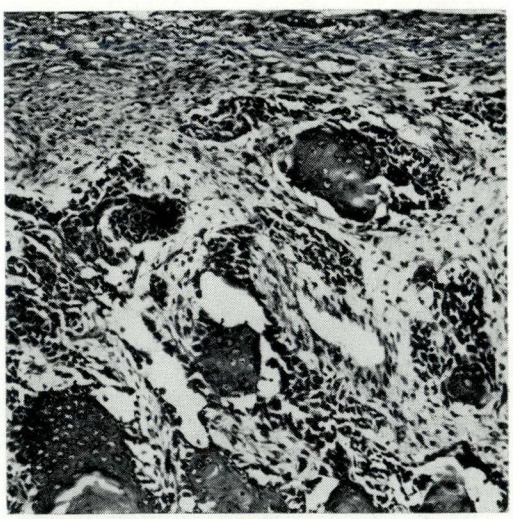

Fig. 2.—Higher magnification of the same lesion as Fig. 1, showing nuclear and cellular pleomorphism. An attempt at horn pearl formation is evident (H & E x 125).

Fig. 3.—Undifferentiated gastric adenocarcinoma in a rat with peptic ulcer (group 6), fed carcinogenic diet for 36 weeks. There is marked hyperplasia of mucosal epithelium with neoplastic glands of varying size penetrating through gastric wall and forming a malignant nodule (H & E x 125).

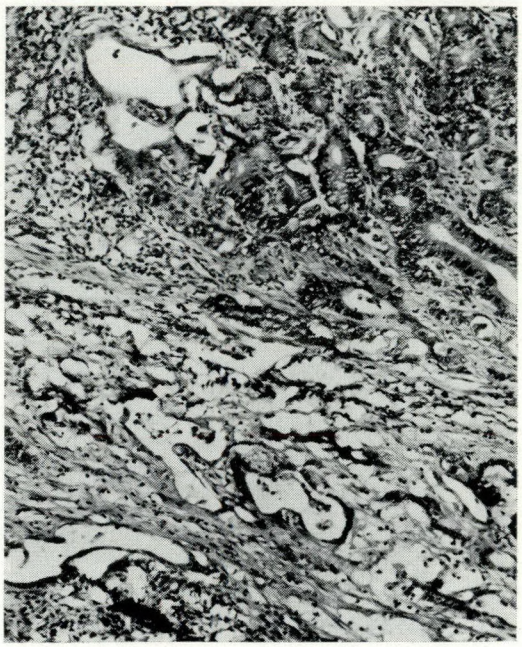

Fig. 4.—Same lesion as in Fig. 3, showing irregular glands lined by atypical epithelium infiltrating through the deep layer of gastric wall (H & E x 125).

An interesting finding was the frequency of gastric mucosal atrophy and intestinal metaplasia in vagotomised rats as compared with those having peptic ulcer only (Table I).

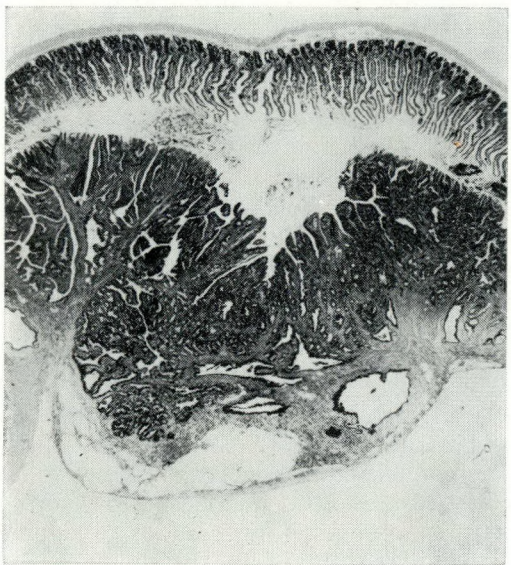

Fig. 5.—Undifferentiated gastric adenocarcinoma in a vagotomised rat (group 7) fed carcinogenic diet for 34 weeks. Marked atypical glandular hyperplasia and invasion by malignant glands may be seen (H & E x 12.5).

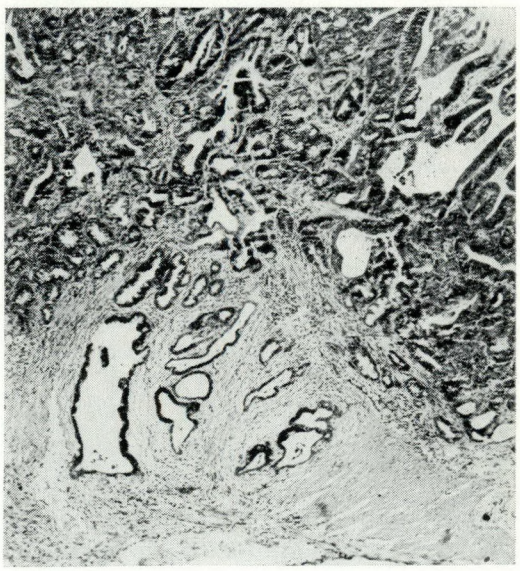

Fig. 6.—Same tumour as in Fig. 5, showing the invasion through all muscular layers of gastric wall (H & E x 50).

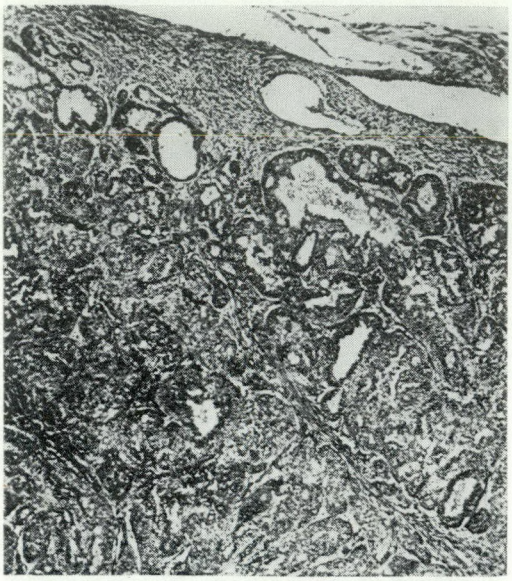

Fig. 7.—Undifferentiated gastric adenocarcinoma in a rat that had both vagotomy and peptic ulcer and was fed for 27 weeks with carcinogenic diet. Invasion to the level of serosa by malignant glandular cells may be noted (H & E x 50).

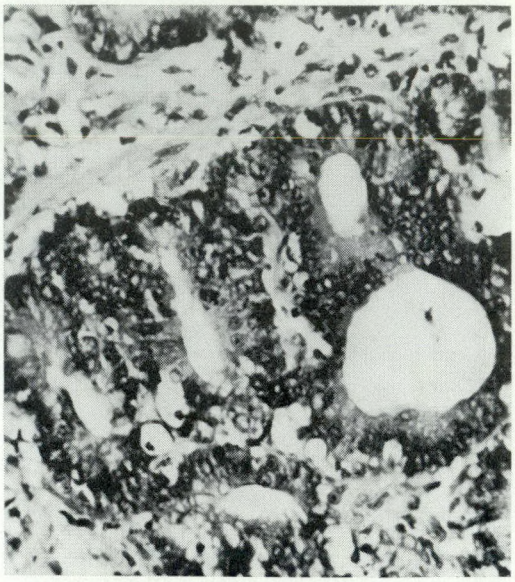

Fig. 8.—Same tumour as in Fig. 7, showing marked nuclear and cellular pleomorphism (H & E x 300).

We have not discussed the histology of gastric mucosa in the first four groups of animals (1-4 in Table II) which received no carcinogen and had no tumours. However, in vagotomised rats (groups 3 and 4), about 10% of stomachs displayed gastric atrophy similar to that found in rats of groups 7 and 8. This finding suggests a possible etiologic relationship between atrophy of glandular mucosa and bilateral truncal vagotomy, atrophy being found between 32 and 46 weeks after denervation of the stomach.

Discussion

This study was concerned with the effect of vagotomy alone or combined with a healing lesion of gastric mucosa on the development of gastric adenocarcinoma in rats.

Vagotomy

As already mentioned, bilateral truncal vagotomy in rats induces complete achlorhydria[4-6] and also impairs gastric circulation and motor function.[5, 20] In vagotomised rats, acute dilatation of the esophagus and stomach is associated with contraction of the cardia and pylorus with resulting delay of gastric emptying.[5, 20] Recent study on ultrastructural changes in the gastric mucosa after truncal vagotomy demonstrated that this procedure causes degeneration of some nerve fibres, vasoconstriction with resulting transitory ischemia, and "aberrations in the substructure of secretory cells" in the gastric mucosa.[22]

Nerve degeneration with resulting cholinergic deficit is responsible for achlorhydria. Ischemia may be responsible for morphologic changes observed in both parietal and chief cells.[22] The present study also shows that gastric mucosal atrophy was frequent in vagotomised rats. It appears, therefore, that vagotomy induces achlorhydria, a variety of motor deficiencies and some abnormalities of gastric circulation. These factors probably decrease the physiologic resistance of gastric mucosa to the oral carcinogen. Ingested carcinogenic substances remain in the stomach for several hours, because post-vagotomy motor deficiency is associated with a marked delay of gastric evacuation. In humans exposed to chronic ingestion of

carcinogen in food,[23] delay in gastrointestinal passage and evacuation of digested food enhance the malignant potential of such a diet. Our present food is contaminated by various preservatives or substances used in agricultural products and some of these or their metabolites may be carcinogenic. Patients with achlorhydric, atrophic and atonic stomachs appear to be particularly vulnerable if exposed to chronic ingestion of these substances in their food.

The present experiment confirms previous clinical and experimental observations regarding association of achlorhydria with gastric carcinoma.

Injury of Gastric Mucosa

As already mentioned, some peptic ulcers in humans become malignant.[8-11] Previously injured gastric glandular mucosa may become a *locus minoris resistentiae* against a carcinogenic factor ingested with the food. Carcinoma develops sometimes after operation for benign lesions of the stomach, particularly if atrophic gastritis preceded the operation. In these instances malignant changes occur near or at the site of anastomosis.[24] Injury enhances the effects of chemical carcinogen on skin,[25-28] and on gastric mucosa.[7, 10, 12, 24] It appears that the promoting factor in malignant transformation is the intimate contact between basal cells, or young regenerating tissue, and the carcinogen.[10] Such a contact was certainly present between 2,7-DAAF and gastric ulcers in rats used in the present experiment. This contact was enhanced by the particular relationship of 2,7-DAAF to gastric tissue: this carcinogen is known to be selectively deposited in the gastric mucosa of the rat.[21] We have, therefore, a satisfactory explanation for the action of carcinogen on the gastric ulcer: the intimate contact of 2,7-DAAF with exposed basal cells and with healing tissue.

Conclusions

This experiment reproduces certain clinical situations in which the genesis of gastric malignancy has been observed in the past. Carcinogenic substances are present in some food.[23] Most of the stomach resists the carcinogenic action of these substances. Normal motor activity, normal secretion of juice rich in H^+ and pepsin, normal mucus barrier, etc., probably protect gastric mucosa against food carcinogens. An atrophic, achlorhydric and atonic stomach, or a stomach with a surgically produced or spontaneous (ulcer) lesion of the gastric mucosa, is less resistant to carcinogens ingested with food. The results of the present experiment support the above conclusions.

Summary

Carcinogen 2,7-diacetylaminofluorene (2,7-DAAF) was given orally in the daily diet to Buffalo male rats. Before the beginning of this experiment, animals had bilateral vagotomy and/or a gastric glandular ulcer induced by thermocautery. Control, carcinogen-fed rats had no operation. Operated rats were also kept on a normal diet and served as controls; they were not exposed to the carcinogen. Only rats exposed to 2,7-DAAF developed malignant gastric lesions. Gastric malignant tumours were classified as adenocarcinomas and mixed tumours (containing both glandular and epidermoid malignant cells). Gastric tumours were found in 6% of rats having no operation, in 25% of rats with peptic ulcer, in 18% of vagotomised animals and in 18% of rats having both surgical procedures. It was concluded that both vagotomy and the presence of injured gastric mucosa (ulcer) enhanced the malignant transformation of gastric glandular mucosa. Under the conditions of this experiment, vagotomy and the presence of peptic ulcer contributed to the development of gastric adenocarcinoma.

The author wishes to thank Dr. H. Sims, Department of Pathology, University of Alberta, for the diagnostic review of histologic slides and the selection of figures, Professor T. K. Shnitka, Department of Pathology, University of Alberta, for his very valuable advice regarding the evaluation of histologic material and Mrs. T. Hoogen, Mr. P. Kim and Mr. K. Lazenby for their technical assistance.

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Résumé

Le but du présent travail était d'établir si l'achlorhydrie gastrique consécutive à la vagotomie et la présence d'un ulcère gastroduodénal en voie de cicatrisation chez le rat (ou l'un des deux signes) ont pour effet d'influencer la morphologie de la muqueuse gastrique soumise à l'action du carcinogène oral qu'est le 2,7-diacétylaminofluorène. On savait déjà, par des études antérieures, que d'étroites corrélations peuvent exister entre l'achlorhydrie et les néoplasmes malins de l'estomac et entre l'ulcère gastroduodénal et les tumeurs gastriques. Nous avons considéré que la vagotomie était la méthode la plus simple de réaliser l'achlorhydrie et que l'administration d'un carcinogène est la meilleure "méthode clinique" pour l'étude des tumeurs gastriques malignes. Nous avons donc donné per os le carcinogène 2,7-diacétylaminofluorène (2,7-DAAF) à des rats mâles de l'espèce Buffalo qui, avant le traitement, avaient subi une vagotomie bilatérale et un ulcère glandulaire induit par thermocautère (ou l'une des deux opérations). On avait comme témoins des rats recevant le carcinogène, mais qui n'avaient pas été opérés. Servaient également

de témoins, des rats opérés, mais dont le régime alimentaire était normal et exempt de carcinogène. Seuls les rats recevant le 2,7-DAAF ont présenté des lésions gastriques malignes. Ces tumeurs ont été classifiées en adénocarcinomes et tumeurs mixtes, ces dernières ayant des cellules malignes, à la fois glandulaires et épidermoïdes. Des tumeurs gastriques ont été découverts chez 6% des rats non opérés, chez 25% des animaux souffrant d'ulcère gastroduodénal, chez 18% des animaux vagotomisés et chez 18% des rats ayant subi les deux opérations. Nous avons donc conclu que la vagotomie et la présence d'une lésion de la muqueuse gastrique (ulcère) ont favorisé la transformation maligne de la muqueuse glandulaire de l'estomac. Dans les conditions de nos expériences, la vagotomie et la présence d'un ulcère ont contribué au développement d'un adénocarcinome gastrique.

INTERVERTEBRAL NERVE-ROOT IN THE INVESTIGATION OF CHRONIC LUMBAR DISC DISEASE*

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OF every 100 patients who undergo operation for lumbar disc disease with associated back pain and sciatica, 10 will continue to complain of significant pain. A second operation will help five of these but the remainder will still suffer from persistent symptoms.[1] A typical patient in the latter group has had at least one operation, yet still complains of low-back pain and sciatica, often without demonstrable neurologic deficit. In many there are accompanying psychologic factors. Roentgenography, myelography and discography are frequently inconclusive.

In undertaking this study we postulated that if the nerve root mediating the symptoms could be blocked selectively with a local anesthetic agent, the symptoms would be temporarily abolished. Conversely, if an asymptomatic nerve root were blocked, the patient's symptoms would be unrelieved. By selectively blocking two or three roots at their intervertebral foramina we have been able to identify accurately the symptomatic nerve root with relative ease and complete safety. The additional information obtained by the test was useful in the management of 23 patients.

METHODS

The investigation is undertaken only at a time when sciatica is actually present. Immediately before the procedure the examiner assesses the patient's disability, the range of back movement, the degree of straight-leg-raising and the neurologic deficit.

The patient is placed in the prone position under an image intensifier and is then rotated until the intervertebral foramina, vertebral pedicles and facets come into better view. To localize the fourth lumbar nerve root, a lumbar puncture (LP) needle is aimed at the intervertebral foramen (Fig. 1), formed by the fourth and fifth lumbar pedicles

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The authors are indebted to Mrs. M. Reid, Research Editor, Division of Neurosurgery, Toronto General Hospital, for her collaboration.

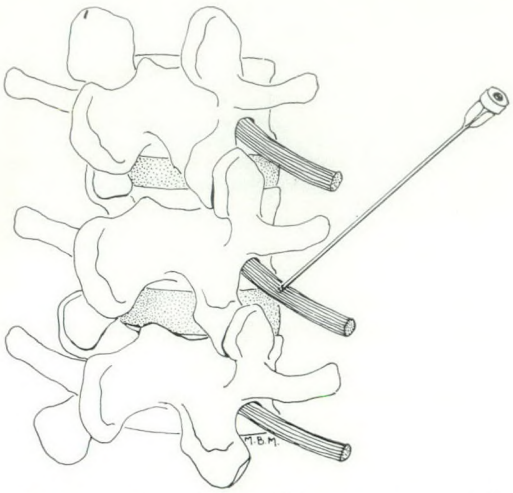

Fig. 1.—LP needle touching a nerve root in the intervertebral foramen.

(a point distal and lateral to the area explored for a disc compressing the fourth lumbar root). Similarly, the fifth lumbar and first sacral roots are identified in their respective foramina.

After the skin has been prepared with an iodine solution, the LP needle is introduced about two inches from the midline and is aimed at the superior level of the intervertebral foramen. The position of the tip of the needle is frequently checked by the image intensifier in posteroanterior and lateral projections. When a nerve root is touched by the LP needle the patient cries out with pain that is like an electric shock and that radiates into the appropriate dermatome. The patient is then asked if the customary type of pain in its usual distribution has been reproduced. Ethodian (ethyl-p-iodophenyl-undecoate) 1 ml., a non-toxic, oily contrast material, is then injected around the nerve root. The contour of the nerve root is followed proximally and distally by the image intensifier (Fig. 2). Finally 1 ml. of procaine is injected around the nerve root. The needle is removed and the patient reexamined. If the symptomatic root is blocked the patient experiences instant relief of all pain. Full trunk flexion and straight-leg-raising are again possible. A few patients have temporary neurologic deficit consisting of numbness in the area supplied by the blocked nerve root. The pain-free state usually wears off within a few hours. If an asymptomatic root is blocked, the patient is not relieved of his pain. We investigate at least one, and sometimes two adjacent roots on separate days to ensure that the symptomatic root is properly identified.

Special care is required to localize the first sacral nerve root. The LP needle must be advanced through the posterior sacral foramen to the anterior sacral foramen where it impinges on the root (Fig. 2). The position of the tip of the needle is checked repeatedly with the image intensifier, to avoid advancing the needle into the pelvis.

Results

The first group we examined comprised 23 patients with 45 separate nerve-root injections. Since then a further 60 patients have been examined, establishing the procedure's merits and

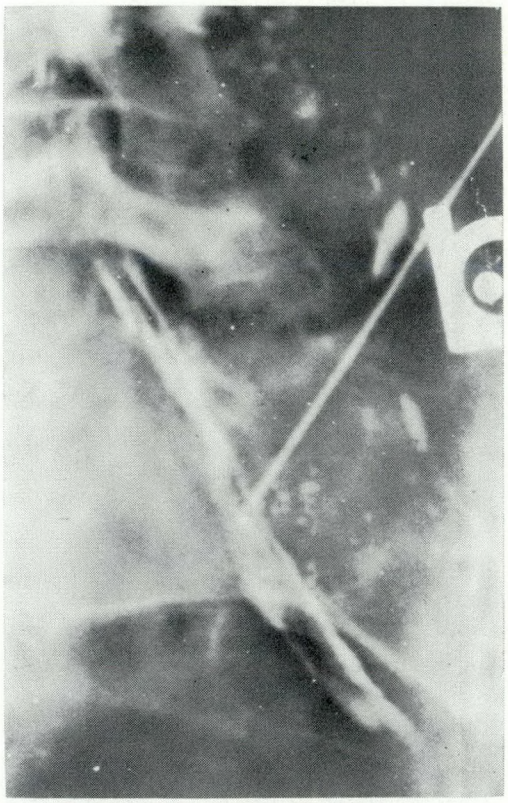

Fig. 2.—The first sacral root outlined by contrast material.

deficiencies. This paper reports only the first group, in whom a detailed correlation was made between the operative findings and the level at which symptoms were produced as indicated by the test.

In 15 patients an operation was performed at the level indicated by the results of the nerve-root block. The operative findings were in agreement with the test findings in 13 patients. We found scarring and fibrous adhesions around the root, bony entrapment at the intervertebral canal, laterally and distally placed sequestrated disc fragments, intradural adhesions, recurrent disc prolapse and, in one case, a foreign body (metallic screw). In five patients the root was firmly adherent to the adjacent pedicle, making an acute angle around the lower pole of the pedicle. In these five cases the offending pedicle was removed (including superior and inferior facets and transverse process), when the root "snapped" into place. In one patient no lesion was found at operation; this was in agreement with the test which had been negative at two levels. Of the two patients in whom the test results and the operative findings did not agree, one had a positive test at the fifth and the other at the third lumbar root. In the latter, a lesion was found at the L3-4 interspace. No lesion was found in the former patient. We do not have an adequate explanation for the discrepant findings, except that they occurred early in the series and may have been owing to inexperience.

The test was useful in seven of the remaining eight patients who were not treated by operation. Three of the eight refused a further operation even though the myelogram and the test indicated a lesion at identical levels. Another three patients improved sufficiently after the test to be discharged from hospital. One patient had relief of pain, regardless of the level injected and whether by procaine or saline, confirming that his complaints had no organic basis. The last patient had had a previous lumbosacral fusion so that the needle could not be advanced into the first sacral foramen.

Discussion

Interpretation of the test requires a discussion of the sinuvertebral nerve, whose significance has been described by many authors.[2-10] Edgar and Nundy[4] dissected the sinuvertebral nerve in a fetus and found that it had a dual origin —a sympathetic component consisting of multiple fine fibres from the corresponding ramus communicans, and a spinal component, originating from one main filament of either the anterior or posterior primary ramus. As many as six separate filaments change direction and pass through the intervertebral foramen and around the base of the pedicle. The distribution of the nerve filaments is mainly segmental but branches ascend and descend for one or two segments. Other branches anastomose with a plexus of nerves situated around and within the posterior longitudinal ligament. Still other branches supply the adjacent dura but leave its dorsal aspect devoid of innervation. The small size of the fibres and the presence of naked nerve endings suggest that this is a pain-conducting system. Because the distribution of the sinuvertebral nerve is diffuse,[11] it is necessary to adhere strictly to the technique as it is described above. The LP needle must be placed exactly in the superior portion of the intervertebral foramen as controlled by the image intensifier and the location of the root must be verified with opaque medium (Fig. 2). The reproduction and the relief of pain must occur at only one level and using only 1 ml. of procaine. The procedure is of great localizing value when all these conditions are met.

The use of local anesthetic agents in the paravertebral region as an aid to the diagnosis and identification of the source of back pain and sciatica is not new. In 1944 White and Gentry[12] described a technique whereby a local anesthetic was injected adjacent to the body of the vertebra and the position was checked radiographically. In 1947 Falconer[13, 14] described a sophisticated procedure by which he placed a needle

lateral to the theca of the root as it passed over the symptomatic disc. He also found that the patient's pain disappeared and straight-leg-raising improved after injecting a local anesthetic. Falconer postulated that this method could be of use in the assessment of the preoperative patient. Later, White[15] again advocated paravertebral procaine and saline injections in the vicinity of the intervertebral foramen as a means of assessing the efficiency of a posterior rhizotomy in intractable neuralgias of non-malignant origin. The present report emphasizes the use of the image intensifier to improve accuracy in locating the symptomatic nerve root.

Of interest was the observation that an anesthetic block distal to the origin of the pain could relieve that pain. The situation is probably analogous to other painful states, e.g. tic douloureux, where distal neurectomies relieve the pain originating from a more proximal source. In 1930 Eccles and Sherrington[16] showed that summation of excitatory effects of a number of afferent nerve fibres is necessary before a discharge is originated. It is probable that a large number of afferents are eliminated by the block so that secondary neurons in the spinal cord are unable to discharge, hence the observation that an anesthetic block of the root could relieve pain originating at a higher level.

Opaque medium (Ethiodan) failed as a reliable indicator of pathologic lesions in the extradural and perineural spaces. On a few occasions structural alterations, such as adhesions, root deformations and disc fragments, were seen on the radiograph and were later confirmed at operation. Most often we were unable accurately to interpret the radiologic aspects of the extradural space, since previous operative dissections and fibrous adhesions had grossly distorted the anatomical relationships.

White[15] has recently rekindled interest in posterior rhizotomy done for intractable non-malignant neuralgias in the lumbosacral region. He found that section of L5 or S1 singly cured eight out of 10 patients who had disabling sciatica due to constricting adhesions. The remaining two were relieved when an adjacent posterior root was also cut. Single posterior rhizotomy was not associated with morbidity, whereas rhizotomy of L5 and S1 produced a temporary proprioceptive impairment at the ankle. Our investigations have allowed us to identify the symptomatic roots with considerable certainty. In a few patients not included in this series we found that we could relieve pain by a single posterior rhizotomy at the level predicted by the test. In these there was no additional neurologic deficit.

There were no complications in the 23 patients. Infection, laceration of nerve roots, cerebrospinal fluid leaks and perforation of a viscus are possible complications. These can be avoided if proper precautions are observed.

Summary

The source of back pain and sciatica persisting after previous operations can be investigated by selectively blocking the nerve roots of the lumbosacral plexus at their intervertebral foramina.

Using the image intensifier, any nerve root (L3-S1) can be accurately identified and outlined with an opaque medium. The procedure is of value in localizing the source of pain, if the pain disappears at only one level when 1 ml. of procaine is injected. It is safe, simple and reliable if it is performed as described in this report.

In 13 of 15 patients the correlation between the findings at operation and the level indicated by the test was accurate. The test was helpful in the management of seven of eight patients in whom no operation was performed.

The management of the patient becomes precise when the test has identified the symptomatic root, since in selected patients a single posterior rhizotomy may be the procedure of choice.

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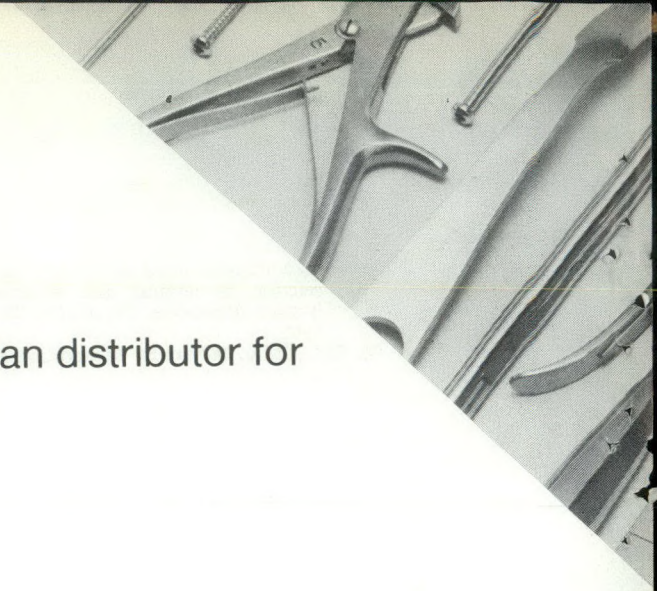
RÉSUMÉ

Il est possible de préciser l'origine d'une lombalgie et d'une sciatalgie qui persistent après des opérations antérieures par un blocage sélectif des racines nerveuses du plexus lombosacré au niveau des trous de conjugaison.

Grâce à l'intensificateur d'image, toute racine nerveuse (L3, L4, L5 et S1) peut être identifiée avec précision et visualisée par une substance de contraste. Cette méthode est précieuse pour localiser l'origine de la douleur, si celle-ci disparaît à un seul niveau quand on y injecte 1 ml de procaïne. Cette méthode est sûre, simple et fiable.

Chez 13 malades sur 15, la corrélation entre les constatations opératoires et le niveau indiqué par l'épreuve a illustré la précision de celle-ci. Ce test a été utile pour traiter sept des huit malades qui n'ont pas été opérés.

Le traitement du malade est grandement facilité quand, grâce au test, on peut identifier la racine symptomatique, étant donné que chez certains malades choisis une simple rhizotomie postérieure peut régler le cas.



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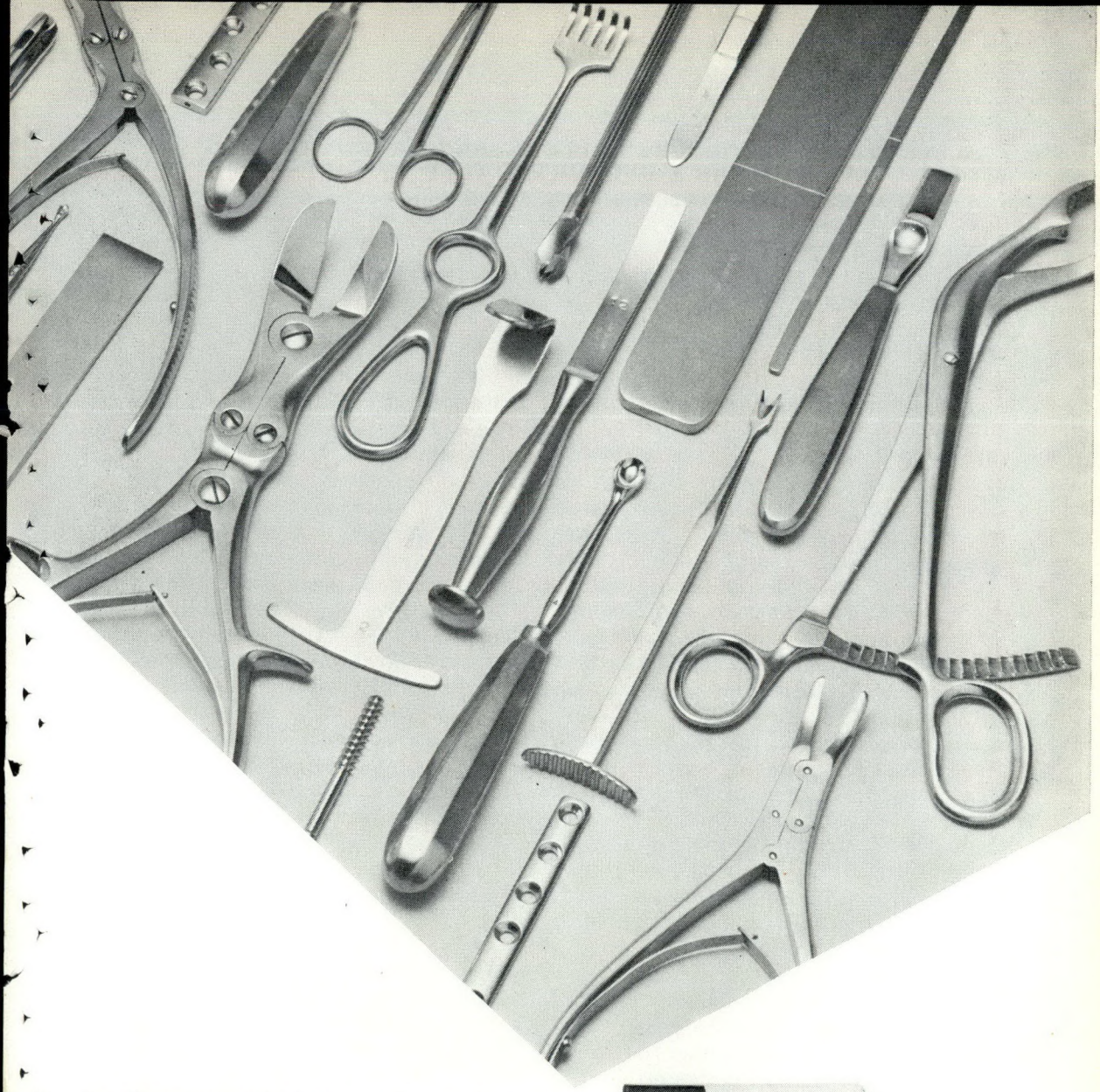

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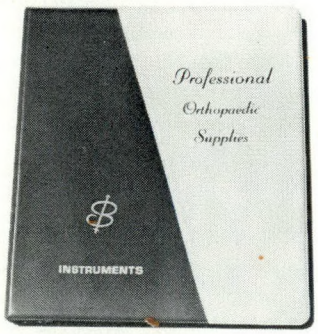

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ACUTE SPINAL CORD INJURY IN PRIMATES PRODUCED BY AN INFLATABLE EXTRADURAL CUFF*

CHARLES H. TATOR, M.D., Ph.D., F.R.C.S.[C],† *Toronto, Ont.*

CURRENTLY a great deal of research is being done on the treatment and pathophysiology of acute spinal cord injury. The stimulus for this effort came primarily from the work by Albin and White and colleagues[1, 2] who showed that the application of local hypothermia by perfusion to the injured spinal cord of animals was an effective treatment for experimentally produced acute spinal cord injury. Injury was inflicted by dropping a weight a known distance on to the exposed thoracic spinal cords of dogs and monkeys. This method of cord injury was developed by Allen[3] in 1911, and other investigators[4, 5] have used it since then. Although it has provided much information about spinal cord trauma, its application to the therapy of spinal cord trauma has been questioned. For example several investigators[6, 7] have been unable to produce a standardized degree of injury with this weight-dropping technique. Although alternative methods for producing experimental spinal cord injuries have been reported, a better model is still needed.

The present report describes a new model of experimental spinal cord injury in which an inflatable Silastic cuff is passed extradurally around the spinal cord of monkeys and rapidly inflated to a given pressure for a given time. The clinical and pathological effects of this circumferential compression injury of the cord are presented.

MATERIAL AND METHODS

Rhesus monkeys weighing 5.5 to 8.5 kg. (obtained from Primate Imports, New York) were cared for in close cooperation with Dr. L. R. Christensen, Director, and Dr. A. Fletch, Veterinarian, of the Division of Laboratory Animal Science at the Medical Sciences Building, University of Toronto. The monkeys were tuberculin-tested on arrival and kept for four weeks before undergoing operation, to allow thorough acclimatization.

Special cages have been developed for the care of the monkeys with spinal cord injuries. These are large enough to allow the monkeys to climb, and have three to four inches of sawdust on the floor. These features are important in the prevention of decubitus ulcers. There are climbing bars and suspended steps in the cages, and when the paraplegic monkeys lie prone on the steps there is sufficient abdominal compression to produce bladder evacuation. In the early postoperative period the monkeys are reluctant to climb, and then any urinary retention is managed by manual abdominal compression or by suspending the animals over the climbing bars and steps.

Operative Procedures

The operations were performed under general anesthesia and aseptic conditions. After sedation with fentanyl citrate-droperidol (Innovar), anesthesia was induced with pentothal and an endotracheal tube inserted; then the monkeys received nitrous oxide and Penthrane and an intravenous saline drip. A laminectomy of T9 and T10 was performed. For five minutes before injuring the cord, during cord injury, and for 10 minutes afterwards the animals were ventilated mechanically, but at other times they were allowed to breathe spontaneously.

A small inflatable Silastic cuff was passed around the cord in the extradural space at T9. This specially designed cuff, obtained from Rhodes Medical Instruments, Woodland Hills, California, is 4 mm. wide, 2 mm. thick and 30 mm. long.

*Supported by the Medical Research Council of Canada and the Canadian Paraplegic Association.

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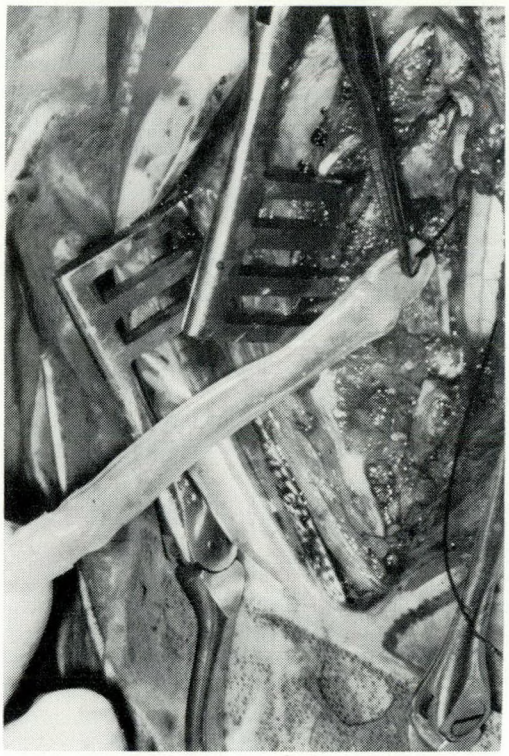

Fig. 1.—(Monkey Z-2). The Silastic cuff is being passed around the spinal cord in the extradural space at T9-10.

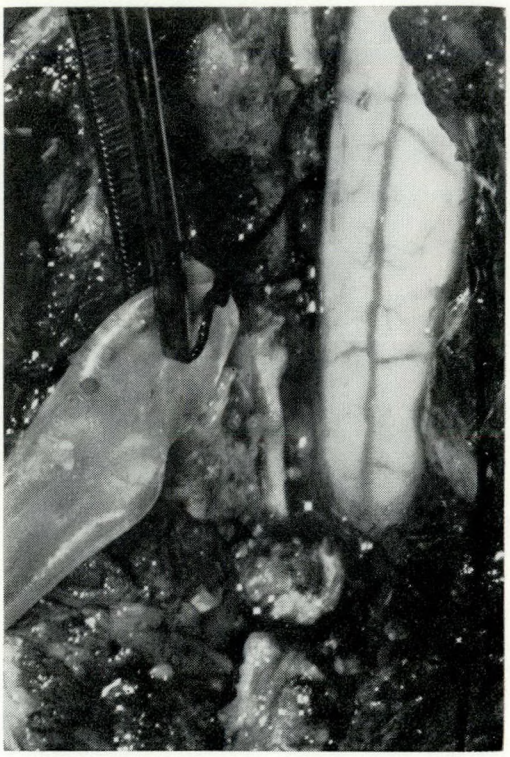

Fig. 2.—Higher power view of Fig. 1. The spinal cord is seen through the transparent dura.

At one end is attached a long Silastic tube through which the cuff can be inflated with air or deflated (Figs. 1 and 2). When not inflated the cuff is elliptical in the transverse plane and after inflation it becomes more circular. To facilitate its passage around the cord, the air contained is first aspirated and this produces flattening of the cuff. After the cuff is in place the vacuum is released. The two arms of the cuff are then approximated over the dorsum of the cord with a cotton ligature in such a way that the cuff completely surrounds the dura and is in contact with it circumferentially (Fig. 3). In order to standardize the placement of the cotton ligature, care is taken to tie it at the level of the dorsal surface of the dura with the ligature in contact with the dura. Although the ligature is tied snugly enough to produce circumferential contact of the cuff with the dura, the lumen of the cuff remains patent. In every instance, immediately after inflation the whole cuff becomes distended throughout its entire length, including the distal tip beyond the cotton tie. Rapid inflation of the cuff is performed manually immediately after placement of the cotton tie, using a Taylor aneroid sphygmomanometer apparatus which permits pressures as high as 400 mm. Hg to be reached in only one to two seconds. After the cuff has been in place for the desired length of time, the cotton tie is quickly divided, the air is evacuated and the cuff removed.

After injury the cord was observed through the dura for three hours and then the muscles, fascia and subcutaneous tissues were closed in separate layers with chromic catgut and the skin with silk sutures. The dura was not opened in these experiments.

Postoperative Assessment of Neurologic Function

Neurologic function was assessed im-

Fig. 3.—(Monkey Z-2). The limbs of the cuff have been approximated and are held in place by a cotton ligature tied flush with the surface of the spinal cord. The cuff is then inflated.

mediately postoperatively, then daily for the first few days and later at least twice weekly. It was graded according to a modification of the Tarlov classification[8] as follows: Grade O—complete paraplegia; Grade 1—flicker of movement; Grade 2—good movement of joints but no weight bearing, walking or use of legs for climbing; Grade 3—use of legs for weight bearing, walking and climbing; Grade 4—normal. Animals were observed for at least 12 weeks postoperatively, and in several instances for much longer periods.

Experimental Protocol

Several studies were made initially to determine the feasibility and design of the compression device. Then a series of monkeys was studied in order to determine the degree of injury necessary to produce immediate and complete paraplegia. This information was necessary so that therapeutic measures could be tested on animals with complete paraplegia, yet with injuries of sufficiently low degree that improvement might occur with treatment. As shown in Table I, progressive increments in pressure and compression times were applied until animals showed immediate complete paraplegia.

Neuropathological Studies

At the end of the 12-week postoperative evaluation period, the whole spinal column was removed and immersed in 10% formalin. After fixation the entire spinal cord was removed and sections were taken for paraffin embedding from the injury site and from the lumbar and cervical regions. Hematoxylin and eosin-stained sections were used for general assessment, Bielschowsky's silver stain for axons, and luxol fast blue for myelin.

Results

It was found that the Silastic cuff could be passed extradurally around the spinal cord without difficulty owing to the presence of abundant extradural fat in the monkey's spinal canal. When flattened by evacuation of its air content the cuff was approximately 1 mm. in thickness and produced minimal distortion of the cord. When the vacuum was released the cord was displaced 2 to 3 mm. posteriorly by the cuff. The cuff has proved to be durable, and the same one was used for all the experiments reported here.

The operative incisions healed well and no wound infections occurred. There were no cerebrospinal fluid fistulas.

Pressure sores developed in many animals and were often aggravated by self-mutilation. In those monkeys which showed some recovery, these sores often healed completely. Assessment of motor function was usually not difficult and the Tarlov classification was a useful method of grading the degree of recovery. Assessment of sensation was difficult because the Rhesus monkeys are aggressive and hostile and resist handling. It was not possible to distract them

TABLE I.—Cuff Pressure and Compression Duration Required to Produce Immediate Complete Paraplegia

Animal	Injury		Result — grade*											
	Cuff pressure (mm. Hg)	Compression duration (min.)	Weeks after operation											
			1	2	3	4	5	6	7	8	9	10	11	12
C-1	Cuff placement only, no inflation		4	4	4	4	4	4	4	4	4	4	4	4
D-1	Cuff placement only, no inflation		4	4	4	4	4	4	4	4	4	4	4	4
E-1	150	2	4	4	4	4	4	4	4	4	4	4	4	4
H-1	150	5	1	1	1	1	1	1	2	2	2	2	2	2
I-1	150	5	1	0	1	1	2	3	3	3	3	3	3	3
F-1	250	5	0	0	0	1	1	1	1	1	1	2	2	2
G-1	250	5	0	0	0	1	2	2	2	2	2	2	2	2
L-1	300	5	0	0	0	1	1	1	1	1	1	1	2	2

*Neurologic deficit graded by a modification of the Tarlov classification — see text.

sufficiently to determine the amount of pain sensation retained.

Amount and Duration of Compression Required to Produce Immediate Complete Paraplegia

Placement of the cuff around the cord in two monkeys, without inflation of the cuff, produced no detectable effect on neurologic function (Table I). When the animals recovered from the anesthetic they were almost immediately able to bear weight, walk and climb. In another monkey compression with the cuff inflated to 150 mm. Hg for two minutes likewise produced no deficit. However, at 150 mm. Hg maintained for five minutes, two other monkeys showed marked neurologic deficit, judged to be Grade 1 in both, immediately postoperatively. One of these animals deteriorated during the second week and became totally paraplegic. Both then gradually improved and at 12 weeks one was Grade 2 and the other Grade 3 (Table I). Two monkeys compressed at 250 mm. Hg for five minutes were Grade 0 immediately postoperatively and both gradually improved to Grade 2 at 12 weeks. Thus in these experiments the cuff pressure and compression duration required to produce immediate complete paraplegia were 250 mm. Hg and five minutes respectively.

Injuries at 350 mm. Hg Pressure for Five Minutes

In 10 monkeys injuries at 350 mm. Hg were inflicted for five minutes, and all the animals were completely paraplegic postoperatively (Table II). During the fourth postoperative week recovery was noted in two monkeys. At 12 weeks, five monkeys remained com-

TABLE II.—Monkeys Injured at 350 mm. Hg for Five Minutes

Animal	Injury		Result — grade*											
	Cuff pressure (mm. Hg)	Compression duration (min.)	Weeks after operation											
			1	2	3	4	5	6	7	8	9	10	11	12
N-1	350	5	0	0	0	0	0	0	0	0	0	0	0	0
P-1	350	5	0	0	0	0	0	0	0	0	0	0	0	0
S-1	350	5	0	0	0	0	0	0	0	1	2	2	2	2
U-1	350	5	0	0	0	0	0	0	0	0	0	0	0	0
V-1	350	5	0	0	0	0	0	0	0	0	0	0	0	0
A-2	350	5	0	0	0	0	1	2	2	2	2	2	2	2
E-2	350	5	0	0	0	1	1	1	1	1	2	2	2	2
F-2	350	5	0	0	0	0	0	1	1	2	2	2	2	2
G-2	350	5	0	0	0	0	0	0	0	0	0	0	0	0
H-2	350	5	0	0	0	1	2	2	2	3	3	3	3	3

*Neurologic deficit graded by a modification of the Tarlov Classification — see text.

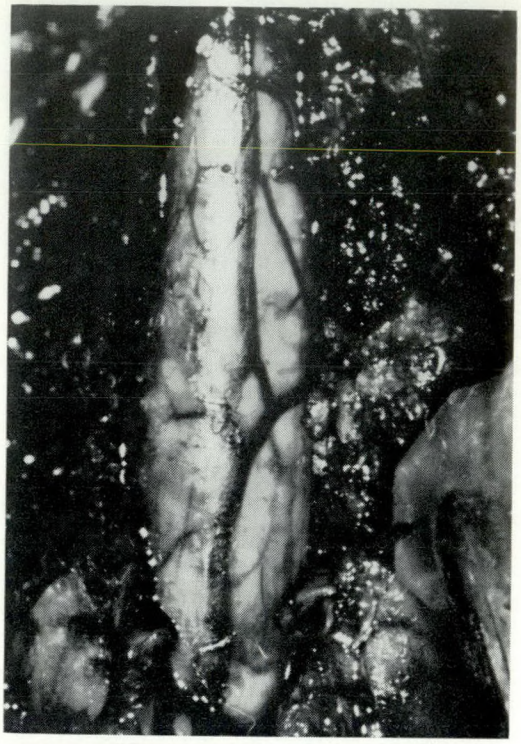

Fig. 4.—(Monkey Z-1). Immediately before injury at 350 mm. Hg for five minutes.

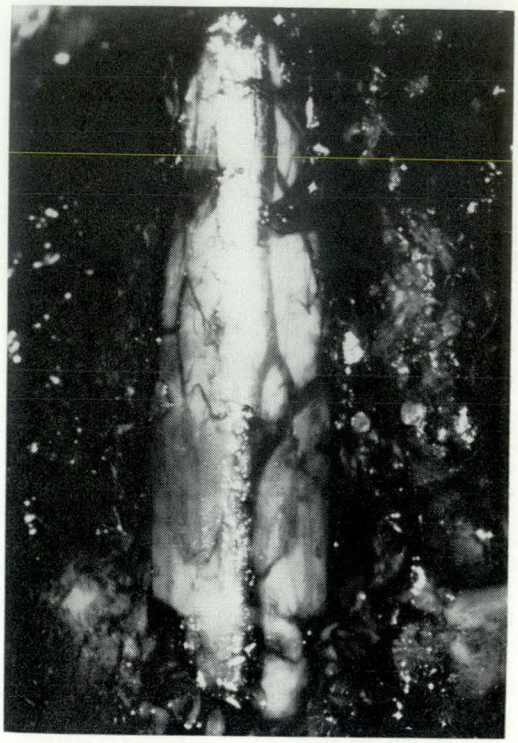

Fig. 5.—(Monkey Z-1). One minute after injury. The vessels have become constricted. The whitish shiny areas are artefact due to light reflection.

pletely paraplegic, four had reached Grade 2 and one had reached Grade 3. None recovered completely.

Gross Observations on the Spinal Cord After Injury

At the lower cuff pressures and compression times minimal changes were noted in the cord at the site of injury. However, at 250 and 350 mm. Hg for five minutes several morphologic changes were usually evident. As soon as the cuff was deflated and removed there was noticeable an hour-glass constriction and a general bluishness of the cord at the injury site. The midline dorsal vein was initially empty but rapidly refilled with blood within one to two seconds after cuff removal. Then the vein often dilated and remained so for several minutes before showing progressive constriction to a smaller calibre than it was before the injury (Figs. 4-6). The blood in the dorsal vein at

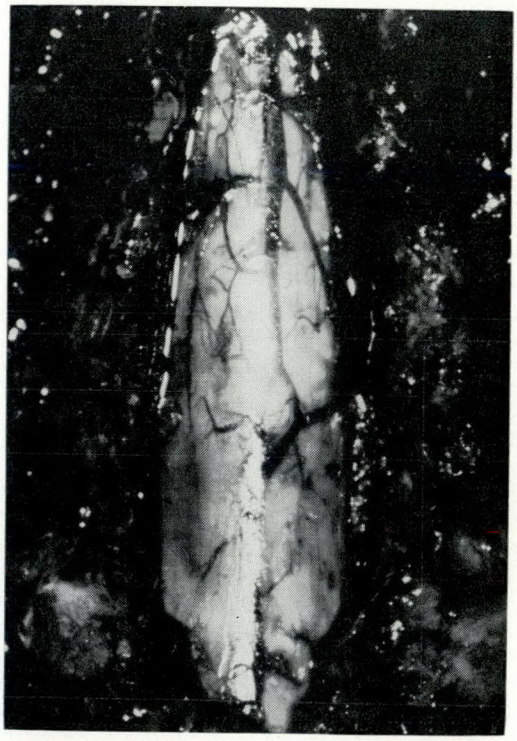

Fig. 6.—(Monkey Z-1). Five minutes after injury. Further vascular constriction has occurred.

the site of injury was often much darker and more venous in appearance after the injury than before, whereas proximal and distal to the injury site the blood in the vein often appeared redder than normal. There were a few subpial petechial hemorrhages in about half of the animals compressed at 350 mm. Hg for five minutes. These were apparent almost immediately after the injury and did not change during the three-hour observation period. At the end of three hours slight swelling of the cord was detected in the animals injured at 350 mm. Hg for five minutes, but the changes in the calibre and colour of the vessels had completely disappeared so that the cords usually appeared completely normal (Fig. 7), except for the slight edema and the few petechial hemorrhages in some of the monkeys.

After 12 weeks, when the cords were removed at autopsy, those compressed at the lower values appeared grossly normal but those compressed at 250 and 350 mm. Hg were flattened in the anteroposterior plane and constricted in the coronal plane. Palpation of the injury site usually revealed softening over a 10- to 15-mm. segment centred about the injury site. When these cords were held up to a strong light there was considerable translucency at the injury site.

Fig. 7.—(Monkey Z-1). Three hours after injury (dura open). The vessels have returned to their normal calibre and the cord appears normal.

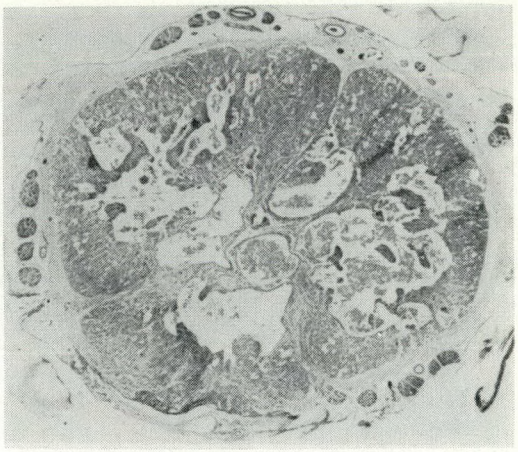

Fig. 8.—(Monkey P-1). Cross section of the spinal cord at the injury site. There is marked destruction of central grey matter with large cavities extending out into the white matter. There is marked gliosis and vacuolation of the remaining white matter (Bielschowsky's stain x 15).

Microscopic Observations

The cords were examined microscopically at least 12 weeks after infliction of the injuries. Cords subjected to pressure less than 250 mm. Hg showed some vacuolation and neuronal loss in the grey matter, and vacuolation and demyelination of the white matter. Most of the cords injured at 350 mm. Hg for five minutes showed large cavities in the central grey matter extending into the white matter for a variable distance (Figs. 8 and 9). In the white matter there was complete demyelination and marked gliosis at the injury site in several animals with only a small number of myelinated fibres remaining in the others. In most there was a marked degree of demyelination of afferent tracts in the cervical region and of the pyramidal tracts in the thoracolumbar region.

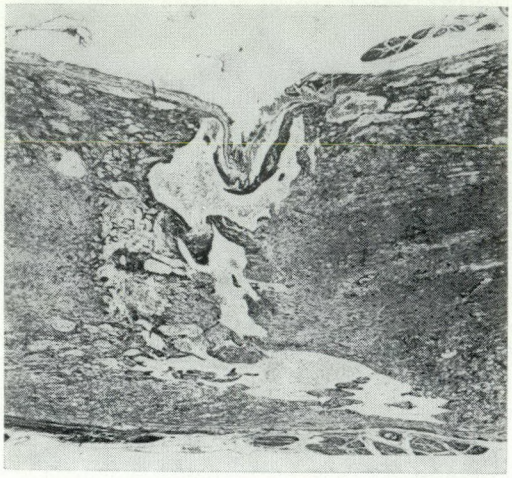

Fig. 9.—(Monkey U-1). Longitudinal coronal section of the spinal cord at the injury site. Marked cavitation of grey and white matter is seen with only a few remaining fibres persisting at the injury site (Bielschowsky's stain x 15.

Discussion

Using the weight-dropping technique, Albin *et al.*[2] found that of 13 untreated monkeys injured by dropping a 20-g. weight 15 cm. (a 300 gram-centimetre-force or gcf) 12 remained completely paraplegic and one recovered a flicker of movement. In contrast, of 14 similarly injured monkeys treated by local perfusion of the exposed cord with normal saline at 2° to 5° C. for three hours beginning four hours after the injury, 13 recovered completely and one almost completely. Unfortunately other investigators have reported less dramatic results with hypothermia, and there has been criticism of the weight-dropping technique. For example, Ducker and Hamit[8] found that some untreated dogs injured by a 375 gcf regained some voluntary movements, while the group whose cords were cooled, although statistically superior to the untreated group, did not recover completely. In addition, cord cooling was not more effective than treatment with dexamethasone. This was disappointing because dexamethasone and other glucocorticoids have been used clinically in spinal cord trauma for several years without definite evidence of major benefit. Kelly *et al.*[9] studied the effects of hypothermia on dogs injured by a 400 gcf and also demonstrated the variability of the results in the hypothermic and non-hypothermic groups. Of the non-hypothermic dogs, two out of 14 recovered full function, while in the dogs in which hypothermia was used complete recovery occurred in three out of 10 treated immediately after injury and in five out of 14 treated after an interval of four hours. Complete paraplegia persisted in five of 14 of the non-hypothermic group, in two of 10 of the immediately hypothermic group and in one of 14 of the delayed hypothermic group. Tomasula *et al.*[6] studied the effects of a 400 gcf on cats and found that two of 17 non-hypothermic cats and five of 11 cats treated with hypothermia immediately after the injury, became ambulatory.

Although the studies of Ducker and Hamit,[8] Kelly *et al.*[9] and Tomasula *et al.*[6] provide additional evidence that hypothermia is of some therapeutic value in this type of cord trauma, hypothermia was not as consistently beneficial as had been reported by Albin's group.[2] These subsequent studies also indicate that the weight-dropping model of experimental cord injury may produce variable results as shown by the recovery of untreated animals whose cords had been injured.

Further evidence that the weight-dropping technique does not injure the cord in a constant, reproducible manner comes from the work of Donaghy and Numoto[7] who used a strain gauge to measure the force exerted on the spinal cord of dogs from a 20-g. weight dropped 30 cm. (600 gcf). The force in 10 animals ranged from 0.9 to 4.9 lbs. with a mean of 3.2. Tomasula *et al.*[6] found considerable variation in the pathological lesions seen in their animals traumatized at 400 gcf and concluded that the technique "cannot be relied upon for consistent production of a standardized lesion in a large series of animals".

Other methods of experimental spinal cord injury have been described. These have included the use of extradural bal-

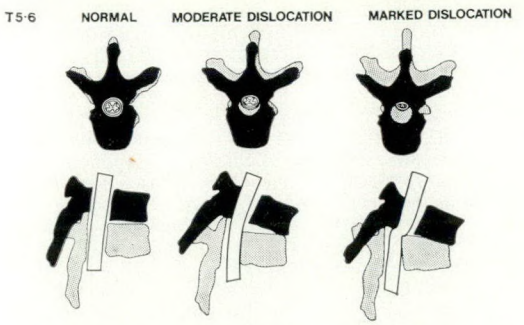

Fig. 10.—Superior and lateral views of the relationships between two thoracic vertebrae and the spinal cord in the normal state (left), after moderate dislocation (middle), and after marked dislocation (right). There is a progressive decrease in the size of the spinal canal. Not only is there compression of the spinal cord between the posterosuperior edge of the body of the vertebra below and the anterior edge of the lamina of the vertebra above as shown in the lateral view, but there is also circumferential compression of the cord as shown in the superior view.

loons and other objects placed along one side of the cord,[10-12] compression of the cord by forceps,[13] and modifications of the weight-dropping technique.[14] Nevertheless, it was felt that there was a need for another model of experimental spinal cord injury for the evaluation of therapeutic measures such as hypothermia. Preferably, the method should simulate the commonest mechanism of spinal cord injury in man, *viz.* the cord compression accompanying dislocation or fracture-dislocation of the spinal column. When the spinal column is dislocated the spinal canal is narrowed and the cord is compressed circumferentially (Fig. 10). The extradurally placed circumferential cuff is an attempt to simulate this mechanism of spinal cord compression.

Several studies have emphasized the importance of changes in the blood supply to the affected cord segment which occur following trauma. These changes result in ischemia of the cord.[11, 12, 15-18] Therefore, it was believed that an experimental model should utilize monkeys because of the similarity in the blood supply to the cord in monkeys and humans.[19] In lower animals the cord is supplied by segmental arteries at all levels, whereas in primates many of these arteries fail to develop with the result that the cord receives only a limited number of medullary arteries.

Our results indicate that extradural circumferential compression of the spinal cord by the inflatable Silastic cuff is a satisfactory method for producing a graded compression injury of the cord. Placement of the cuff in the extradural space is not difficult and the device is sufficiently durable to allow repeated use.

Immediate and complete paraplegia occurred when the extradural cuff was inflated to 250 mm. Hg and left in place for five minutes. However, both monkeys thus injured recovered in time (Table I). At 350 mm. Hg for five minutes, five monkeys remained completely paraplegic throughout the 12-week observation period and five showed some degree of recovery (Table II). None of the monkeys regained normal function and only one was able to use his legs for walking and weight-bearing. It is likely that the variation in response is due to differences in the actual pressure exerted on the cord by the cuff, possibly resulting from the varying spatial relationships between cords of various widths and the cuff. Undoubtedly some variation also arises from unavoidable differences in the tightness with which the limbs of the cuff are apposed by the ligature, and in the available space for the cuff in the extradural plane.

The pathophysiology of the lesions produced by this method is not known with certainty. The two possible mechanisms involved are ischemia and direct compression, but the relative importance of each could not be ascertained. The early changes observed in the veins on the surface of the cord at the site of injury suggest a degree of stagnation of blood flow followed by vasoconstriction. The extreme softening, cavitation, and demyelination at 12 weeks seen in animals injured at 350 mm. Hg for five minutes indicate that a considerable degree of necrosis occurred. Even in those animals recovering some function there was

marked cavitation and demyelination. One of the striking findings was the similarity in the degree of destruction of cord tissue among the animals injured at 350 mm. Hg pressure for five minutes. It was remarkable how much necrosis was present, even in those animals which had some recovery.

Conclusion

An experimental model of spinal cord injury has been developed which consists of circumferential compression of the cord by an inflatable extradural cuff. The model attempts to produce compression of the cord similar to that which accompanies dislocation of the spine. The clinical and pathological effects of this injury have been shown. It is considered that the model is sufficiently reliable for use in controlled studies of the effectiveness of various therapeutic measures for spinal cord injuries.

The author expresses his appreciation to Mrs. M. Marmash, Mrs. F. Crawford, Miss M. Dubois and Mr. M. Bovell for technical assistance, to Miss P. Davidson for the art work, to Mr. J. Kozie for the photographs and to Drs. L. R. Christensen and A. Fletch for assistance with the management of the monkeys.

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Résumé

L'auteur a mis au point une méthode permettant de provoquer artificiellement des lésions médullaires chez les singes et qui a pour but de reproduire la lésion compressive aiguë de la moelle épinière chez l'homme. Cette méthode utilise un petit manchon gonflable, fait de Silastic dont on entoure la moelle dans l'espace extradural au niveau T-9 et dont assure le maintien en place et le contact circulaire avec la dure-mère. Ce manchon est rapidement gonflé à une pression donnée pendant une période donnée.

Une pression de 150 mm Hg maintenue pendant 5 minutes n'a pas entraîné une paraplégie complète, tandis qu'une compression de 250 mm Hg pendant le même laps de temps a provoqué

immédiatement une paraplégie complète qui s'est révélée réversible avec le temps. Les lésions médullaires causées par une compression de 350 mm Hg durant 5 minutes chez 10 singes ont causé une paraplégie complète immédiatement après l'opération. Cinq de ces animaux sont restés complètement paraplégiques pendant la totalité de la période d'observation de 12 semaines après la création de la lésion et les cinq autres se sont remis partiellement.

Les modifications vasculaires observées sur la surface de la moelle consistaient en dilatation et stase veineuse, suivies de constriction. Les lésions les plus sévères consistaient en hémorragies pétéchiales situées sous la pie-mère. L'examen pathologique de la moelle après 12 semaines, révéla la présence d'une démyélinisation et de vacuoles dans les lésions bénignes évoluant vers une cavitation et un infarctus complet aux niveaux plus élevés de compression.

L'auteur estime que ce modèle expérimental sera utile pour évaluer diverses méthodes thérapeutiques des lésions aiguës de la moelle épinière.

BOOK REVIEWS

THE CARE OF MINOR HAND INJURIES. 3rd ed. Adrian E. Flatt. 293 pp. Illust. The C. V. Mosby Company, St. Louis, 1972. $22.60.

In the third edition of this book written by the Professor of Orthopedic Surgery at the University of Iowa, the author emphasizes that minor hand injuries are not to be considered trivial but rather demanding of care and expertise. The organization of the material is exemplary and provides ready access to specific areas and injuries.

The illustrations are excellent and a credit to Mr. Hage, the artist. The radiographs are well reproduced.

The book does not cover the treatment of hand injuries in their entirety, but focuses on the author's specific purpose, namely, to emphasize appropriate treatment for the acutely compromised hand with a "minor injury".

The clarity of presentation and generally conservative tone make this a worthwhile text to be consulted in every emergency. While it could be considered unsophisticated, this is hardly a criticism, but rather a recommendation.

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INSTRUCTIONAL COURSE LECTURES. Volume XXI, 1972. The American Academy of Orthopaedic Surgeons. 332 pp. Illust. The C. V. Mosby Company, St. Louis, 1972. $22.50.

This volume of Instructional Course Lectures maintains the usual high standard of publications produced by the American Academy of Orthopaedic Surgeons. The papers are all well written and the illustrations throughout are of a very high quality indeed.

The subjects covered in this year's volume are prevention and therapy of pyogenic infections in orthopedics; management of chronic orthopedic pain problems; introduction to clinical electromyography; humeral head defects associated with shoulder dislocations; Sprengel's deformity; treatment of the stiff hand and flexible implant arthroplasty in the fingers; hysterical backache; the long-term management of thoracolumbar fractures and fracture-dislocations; pathology and current treatment concepts of cervical spine injuries; surgery of the cervical spine; management of soft cervical disc herniation; diagnostic approach to the dysmorphic child; prenatal development of the human hip joint; congenital dislocation of the hip in infancy; diagnosis, pathology, etiology, mechanism and incidence of slipped capital femoral epiphysis; treatment of minimally slipped upper femoral epiphysis; treatment of severely slipped upper femoral epiphysis by wedge osteotomy, trochanteric osteotomy and by osteoplasty and epiphyseodesis; treatment of acute slipped upper femoral epiphysis; chrondrolysis after epiphyseolysis; implant arthroplasty in disabilities of the great toe; arthrodesis for hallux valgus; treatment of hallux valgus in rheumatoid arthritis; Mitchell osteotomy-bunionectomy for hallux valgus; modified Akin procedure for the correction of hallux valgus; hallux valgus in the adolescent; sling procedure in the teenager; modified Joplin sling procedure for splayfoot; Le-Lièvre bunion operation and slide preparation.

This book will be of particular value to orthopedic residents during their training. A copy should be on the bookshelf of every orthopedic department library. It will also be of considerable value as a reference book for the use of practising orthopedic surgeons and those engaged in the teaching of this specialty.

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MANAGEMENT OF EMERGENCIES IN THORACIC SURGERY. 2nd ed. John Borrie. 478 pp. Illust. Appleton-Century-Crofts, Education Division, Meredith Corporation, New York, 1972. $17.00.

It is some 15 years since the first edition of this book was published. This second edition was received with some interest by this reviewer and is a substantial improvement over the first. The text has been expanded; the illustrations are again of high quality but the references are by no means up-to-date. The book reflects the personal experience of an eminent thoracic surgeon.

Dr. Borrie does not discuss or even mention postoperative tracheal stenosis as a complication. In his management of fractured ribs, one would suggest that a continuous epidural anesthetic is far superior to intermittent injections to block the intercostal nerves. In rupture of the bronchus it is never too late to perform repair and some degree of pulmonary

(Continued on Adv. page 31)

(Continued from page 232)

function can be obtained even when restoration of bronchial continuity is delayed until many years later. The mention of a Heimlick valve in the management of spontaneous pneumothorax would have been welcome for its use can save many hospital days. The underwater seal drainage recommended after pneumonectomy is quite unnecessary and can lead to serious complications from mediastinal shift. It is encouraging to find that someone else believes that preoperative digitalization before major thoracotomy in older people is of benefit. It is interesting to see osteoplastic thoracoplasty mentioned as a procedure, even though this technique is now seldom used. In the discussion of bleeding esophageal varices, one would have liked to see mention of emergency portocaval shunts because such a technique does have a place in their management. The sections on heart emergencies could have been expanded and in traumatic rupture of the thoracic aorta, one would have preferred to see femorofemoral bypass advocated to protect the spinal cord in place of the procedures mentioned in the book.

On the whole, despite the above remarks, this is an excellent text which outlines the management of many of the emergencies seen in thoracic practice. It can be recommended without reservation to anyone interested in this field and should be available in every medical library and emergency department for the use of medical students, residents and interns. It also contains many helpful hints for the mature thoracic surgeon.

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SYMPOSIUM ON AESTHETIC SURGERY OF THE FACE, EYELID, AND BREAST. Vol. IV. Edited by Frank W. Masters and John R. Lewis. 222 pp. Illust. The C. V. Mosby Company, St. Louis, 1972. $37.50.

This book presents a symposium organized by the Educational Foundation of the American Society of Plastic and Reconstructive Surgeons and the American Society for Aesthetic Plastic Surgery which was held in Phoenix, Arizona, in November 1970. It consists of four basic divisions containing contributions from 38 authors.

The opening section is an introduction to the subject of aesthetic surgery and discusses the preoperative assessment of the patient and the socioeconomic considera-

(Continued on Adv. page 34)

You won't need 8 grams/day I.V. for most staphylococcal pneumonias, but that's how high you can go with Lincocin when serious infections are life-threatening

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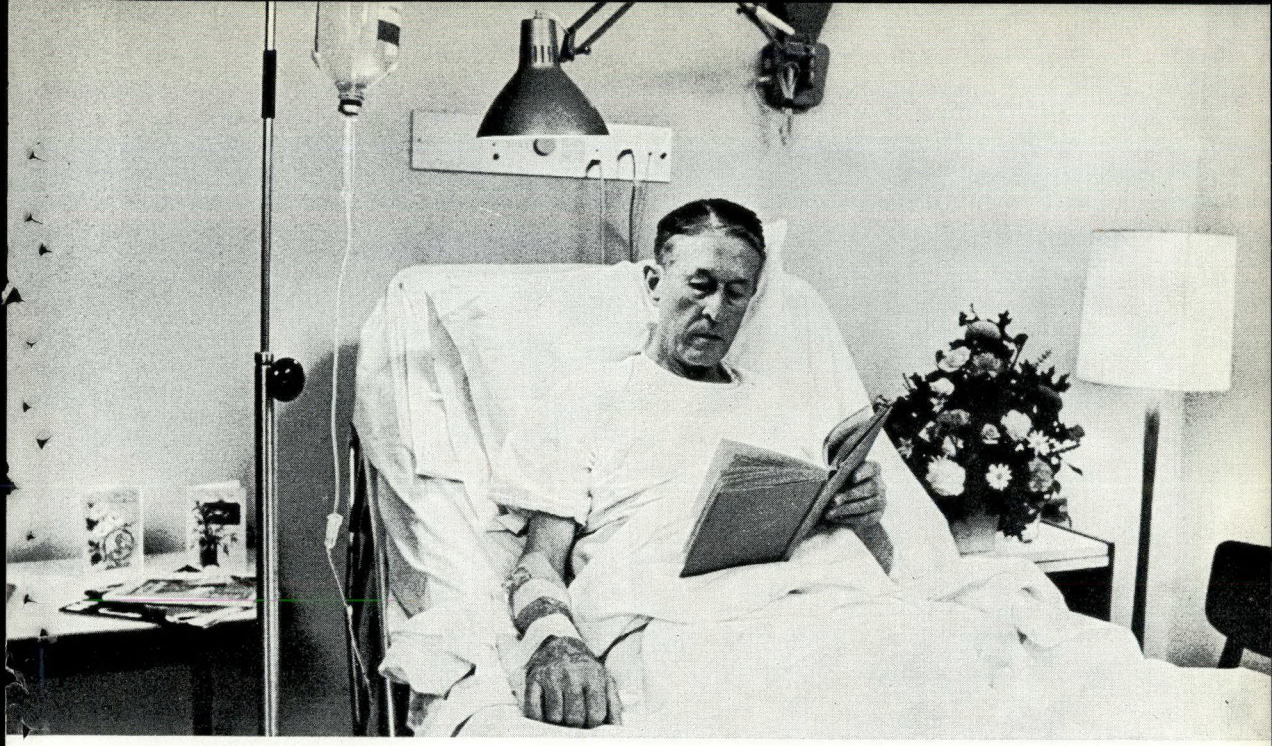

1.2 to 1.8 grams/day is usual I.V. dosage: A clinically useful feature of intravenous therapy with Lincocin is its broad dosage range, from 1.2 to 8.4 grams/day. However, most patients with uncomplicated pneumonias respond satisfactorily to 0.6 grams every 8 to 12 hours.

Up to 8.4 grams/day for life-threatening infections: For more serious infections, the usual Lincocin doses may have to be increased. Doses as high as 8.4 grams per day for 7 days have been given by I.V. infusion to adults.

See "dosage" below for administration recommendations.

Effective Gram-positive antibiotic: Lincocin is highly effective in respiratory, skin, soft tissue and bone infections caused by pneumococci, streptococci and staphylococci, including penicillin-resistant strains.

Well tolerated systemically: Hypersensitivity reactions to Lincocin are rare. Lincocin does not share antigenicity with the penicillins or cephalosporins and has proved valuable in treating patients sensitive to these compounds.

Well tolerated at the injection site: Intravenous infusions of Lincocin have not produced local irritation or phlebitis, when given as recommended. Lincocin is also well tolerated when given by intramuscular injection.

Indications: Infections caused by Gram-positive organisms which are susceptible to the action of Lincocin, particularly staphylococci (including penicillinase-producing staphylococci), streptococci, and pneumococci. Not active against Streptococcus faecalis, yeasts, or Gram-negative organisms including N. gonorrhœae and H. influenzae.

Dosage and Administration:

Intravenous – *Adults:* 600 mg-2100 mg (2 ml-7 ml) intravenously every 8 to 12 hours. For intravenous administration, Lincocin should be added to 250 ml or more of 5% glucose in water or normal saline and given as an infusion over a period of 30 to 120 minutes.

When doses of 4 grams or more are given, Lincocin should be diluted in 500 ml of fluid and administered at a rate not to exceed 100 ml per hour.

Doses as high as 8.4 grams per day for 7 days in four divided doses of 2100 mg given in an infusion of 250 ml of normal saline over a period of 120 minutes were well tolerated in normal adult volunteers.

Children over one month of age: 10 to 20 mg/kg/day (5 to 10 mg/lb/day) divided into 2 or 3 doses and given as an infusion at 8- to 12-hour intervals. The method of administration is the same as noted above for adults.

For serious infections, these doses may have to be increased.

Intramuscular – *Adults:* Mild infections – 600 mg (2 ml) intramuscularly every 24 hours. Severe infections – 600 mg (2 ml) intramuscularly every 12 hours or more often.

Children over one month of age: Mild infections – one intramuscular injection of 10 mg/kg (5 mg/lb) every 24 hours. Severe infections – one intramuscular injection of 10 mg/kg (5 mg/lb) every 12 hours or more often.

Cautions: Generally well tolerated. With administration of Lincocin oral preparations, gastrointestinal side effects have been encountered such as loose stools or diarrhœa, nausea, vomiting, and abdominal cramps. Other minor side effects have been observed infrequently. Side effects such as neutropenia, leukopenia, agranulocytosis and hypersensitivity reactions have been observed on rare occasions. Should not be used in patients sensitive to clindamycin.

Pending further clinical experience, Lincocin is not recommended in the newborn, in the prophylaxis of a recurrence of rheumatic fever, and in patients with pre-existing kidney, liver, endocrine, or metabolic diseases.

Since safe conditions for the *parenteral* use of Lincocin in pregnancy have not been established, its use in such patients should involve careful consideration of expected benefits and possible risks.

Detailed information is available on request.

Availability:

Lincocin Sterile Solution: Each ml contains lincomycin hydrochloride equivalent to 300 mg lincomycin base, in 2 ml and 10 ml vials.

Also available for oral administration:

Lincocin Capsules: Each capsule contains lincomycin hydrochloride monohydrate equivalent to 500 mg lincomycin base, in bottles of 12 and 100.

Lincocin Syrup: Each 5 ml (teaspoonful) contains the equivalent of 250 mg lincomycin base (from lincomycin hydrochloride monohydrate). Bottles of 30 ml (with calibrated dispenser), 60 ml, and 16 oz.

Upjohn

725 REGISTERED TRADEMARK: LINCOCIN CE 6597.1

PMAC

THE UPJOHN COMPANY OF CANADA/DON MILLS, ONTARIO

(Continued from page 31)

tions. Some of the views expressed will find a varying degree of application depending upon the pattern of the plastic surgeon. Many of these ideas do not relate to the Canadian situation.

Part II is introduced by the history of the surgery of the ageing face and includes an excellent bibliography on the subject. Individual chapters describe the anatomy, surgical techniques and complications of rhytidectomy. Two chapters on chemical face peel are included in this section. The excellent illustrations complement the well-organized text, and most plastic surgeons will find invaluable technical ideas and innovations.

Part III is devoted to the aesthetic surgery of the eyelids. The final section describes aesthetic surgery of the breast. There are chapters on the radiographic and thermographic examination of the breast and on the treatment of micromastia by various implants, with descriptions of their contraindications and complications, as well as of their advantages. The most common and popular methods of correction of the hypertrophic and ptotic breast are described.

This is a well-illustrated, well-written textbook on aesthetic surgery with an excellent bibliography which should have great appeal to the young plastic surgeon as well as to those established in the specialty.

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SYMPOSIUM ON MYELOMENINGOCELE, Hartford, Connecticut, November, 1970. American Academy of Orthopaedic Surgeons. 317 pp. Illust. The C. V. Mosby Company, St. Louis, 1972. $25.20.

This publication contains the series of papers delivered at the Symposium on Myelomeningocele held in Hartford, Connecticut, November 1970, under the auspices of the Committee on the Care of the Handicapped Child of the American Academy of Orthopaedic Surgeons.

The book provides an orderly approach to the embryology, pathology and clinical evaluation of children born with myelomeningocele and related anomalies. The publication is particularly well planned in presenting the complications that accompany myelomeningocele. Sections dealing with hydrocephalus, urinary tract, lower bowel, orthopedic and developmental problems, infection and the future of these patients are presented. Multiple authors from medical specialties and

allied health professions concerned with the care of these patients describe an up-to-date team approach to the philosophy, treatment methods and social problems which are important in the management of patients with this condition.

This book should be of considerable interest to both trainees and qualified members of the specialties concerned. Medical and paramedical personnel involved in the care of these handicapped children will find interesting the emphasis on the updated team approach to their problems.

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SYNOPSIS OF GROSS ANATOMY. 2nd ed. John B. Christensen and Ira Rockwood Telford. 304 pp. Illust. Harper & Row, Publishers, Inc., New York, 1972. $10.95. Paperbound.

As a synopsis, this new book does selectively shorten the enormous subject matter of gross anatomy. Its regional presentation is adequate but not outstanding among the many books currently attempting to find the elusive holy grail of "core" anatomical material. Especially helpful to the beginner is its introductory thumbnail sketch of useful principles and general systemic concepts. Here, I believe, lies its clearest asset. Its major fault is one that is common to most anatomical texts, however well intended, namely the inability to distinguish by differences of type items of great importance from those of little or no consequence. At very least, the use of italics in contrast to heavy type should serve to demote the insignificant material from the significant—to separate "obturator artery" (page 161), for example, from its "anterior branch" and its "posterior branch", or "intervertebral discs" (page 208) from "intertransverse ligaments", and so on.

No one seriously disputes the plain fact that a knowledge of gross anatomy is, today as in the past, the absolute, non-negotiable basis of the study and practice of medicine. But in this reviewer's opinion, it is all the more urgent today for authors of anatomy texts to indicate by their careful selection and discriminating presentation their growing concern not simply to condense but to help the eager, beleaguered student of anatomy learn efficiently and effectively, both according to his needs and to his interests.

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